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DB Name	Query	Hit Count	Set Name
USPT,JPAB,EPAB,DWPI	l17 and l18	7	L19
USPT,JPAB,EPAB,DWPI	l11 same (bound or complexed)	12	L18
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USPT,JPAB,EPAB,DWPI	l11 and (bound or complexed)	105	L15
USPT,JPAB,EPAB,DWPI	l11 and (free or unbound or uncomplexed)	111	L14
USPT,JPAB,EPAB,DWPI	l11 and l5	6	L13
USPT,JPAB,EPAB,DWPI	l11 and l4	7	L12
USPT,JPAB,EPAB,DWPI	l1 same (antibod$3 or receptor$1 or probe$1 or anti-troponin$1 or antitroponin$1)	162	L11
USPT	4727019.pn.	1	L10
USPT,JPAB,EPAB,DWPI	l1 and l6	5	L9
USPT,JPAB,EPAB,DWPI	l1 and l5	6	L8
USPT,JPAB,EPAB,DWPI	l1 and l4	7	L7
USPT,JPAB,EPAB,DWPI	l1 and l2	5	L6
USPT,JPAB,EPAB,DWPI	ternary same ((troponin c) or ctnc or tnc) same ((troponin t) or ctnt or tnt)	6	L5
USPT,JPAB,EPAB,DWPI	binary near10 ((troponin c) or ctnc or tnc)	8	L4
USPT,JPAB,EPAB,DWPI	ternary near10 ((troponin c) or ctnc or tnc) near 10 ((troponin t) or ctnt or tnt)	0	L3
USPT,JPAB,EPAB,DWPI	binary near5 ((troponin c) or ctnc or tnc)	6	L2
USPT,JPAB,EPAB,DWPI	(troponin i) or tni or ctni	553	L1

(FILE 'HOME' ENTERED AT 13:32:07 ON 18 SEP 2001)

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS' ENTERED AT 13:32:26 ON 18 SEP 2001

L1 8664 S (TROPONIN C) OR CTNI OR TNI
L2 105 S BINARY (5A) ((TROPONIN C) OR CTNC OR TNC)
L3 63 S TERNARY (10A) ((TROPONIN C) OR CTNC OR TNC) (10A) ((TROPONIN
L4 105 S L1 (6P) L2
L5 63 S L1 (6P) L3
L6 964 S L1 (6P) (ANTIBOD? OR RECEPTOR? OR PROBE? OR ANTITROPONIN OR A
L7 45 S L6 AND L4
L8 20 S L6 AND L5
L9 15 DUP REM L7 (30 DUPLICATES REMOVED)
L10 6 DUP REM L8 (14 DUPLICATES REMOVED)
L11 189 S L1 (6A) (BOUND OR COMPLEX?) AND (UNBOUND OR FREE OR UNCOMPLEX
L12 61 S L11 AND L6
L13 19 DUP REM L12 (42 DUPLICATES REMOVED)
L14 1 S L1 AND (SENSITIV? (3A) ANTIBOD?)

L9 ANSWER 4 OF 15 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999326171 MEDLINE
DOCUMENT NUMBER: 99326171 PubMed ID: 10395953
TITLE: Conformational changes induced in troponin I by interaction
with troponin T and actin/tropomyosin.
AUTHOR: Tao T; Gong B J; Grabarek Z; Gergely J
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, MA 02114, USA.
CONTRACT NUMBER: AR21673 (NIAMS)
RR11301 (NCRR)
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1999 Jul 8) 1450 (3) 423-33.
Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990817

AB Troponin I (**TnI**) is the inhibitory component of the striated muscle Ca2+ regulatory protein troponin (Tn). The other two components of Tn are **troponin C** (TnC), the Ca2+-binding component, and troponin T (TnT), the tropomyosin-binding component. We have used limited chymotryptic digestion to **probe** the local conformation of **TnI** in the free state, the **binary TnC*** **TnI** complex, the ternary **TnC***. **TnI***TnT (Tn) complex, and in the reconstituted Tn*tropomyosin*F-actin filament. The digestion of **TnI** alone or in the TnC***TnI** complex produced initially two major fragments via a cleavage of the peptide bond between Phe100 and Asp101 in the so-called inhibitory region. In the ternary Tn complex cleavage occurred at a new site between Leu140 and Lys141. In the absence of Ca2+ this was followed by digestion of the 1-140 fragment at Leu122 and Met116. In the reconstituted thin filament the same fragments as in the case of the ternary complex were produced, but the rate of digestion was slower in the absence than in the presence of Ca2+. These results indicate firstly that in both free **TnI** and **TnI** complexed with TnC there is an exposed and flexible site in the inhibitory region. Secondly, TnT affects the conformation of **TnI** in the inhibitory region and also in the region that contains the 140-141 bond. Thirdly, the 140-141 region of **TnI** is likely to interact with actin in the reconstituted thin filament when Ca2+ is absent. These findings are discussed in terms of the role of **TnI** in the mechanism of thin filament regulation, and in light of our previous results [Y. Luo, J.-L. Wu, J. Gergely, T. Tao, Biochemistry 36 (1997) 13449-13454] on the global conformation of **TnI**.

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also in the region that contains the 140-141 bond. Thirdly, the 140-141 region of **TnI** is likely to interact with actin in the
reconstituted thin filament when Ca2+ is absent. These findings are discussed in terms of the role of **TnI** in the mechanism of thin filament regulation, and in light of our previous results [Y. Luo, J.-L. Wu, J. Gergely, T. Tao, Biochemistry 36 (1997) 13449-13454] on the global conformation of **TnI**.

L9 ANSWER 5 OF 15 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1999132222 MEDLINE
DOCUMENT NUMBER: 99132222 PubMed ID: 9931043
TITLE: Determination of cardiac troponin I forms in the blood of patients with acute myocardial infarction and patients receiving crystalloid or cold blood cardioplegia.
AUTHOR: Giuliani I; Bertinchant J P; Granier C; Laprade M; Chocron S; Toubin G; Etievent J P; Larue C; Trinquier S
CORPORATE SOURCE: Sanofi Diagnostics Pasteur, Z.A. Leopha Rue d'Italie, 69780
Mions, France.
SOURCE: CLINICAL CHEMISTRY, (1999 Feb) 45 (2) 213-22.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990223

AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three
immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human **cTnI**. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb.
The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and 10 serum samples from patients receiving cold blood cardioplegia during heart surgery were assayed by the three two-site immunoassays. We confirmed that **cTnI** circulates not only in free form but also complexed with the other troponin components (TnC and cTnT). We showed that the predominant form in
blood is the **cTnI-TnC binary** complex (IC).
Free **cTnI**, the **cTnI**-cTnT **binary** complex, and
the cTnT-**cTnI-TnC** ternary complex were seldom present,
and when present, were in small quantities compared with the binary complex IC. Similar results were obtained in both patient populations studied. These observations are essential for the development of new immunoassays with improved clinical sensitivity and for the selection of an appropriate **cTnI** primary calibrator.
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L9 ANSWER 15 OF 15 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 77071481 MEDLINE
DOCUMENT NUMBER: 77071481 PubMed ID: 187592
TITLE: Effect of Ca2+ binding on troponin C. Changes in spin label
mobility, extrinsic fluorescence, and sulfhydryl reactivity.
AUTHOR: Potter J D; Seidel J C; Leavis P; Lehrer S S; Gergely J
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1976 Dec 10) 251 (23) 7551-6.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197702
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19970203
Entered Medline: 19770216

AB The Ca2+ binding component (TnC) of troponin has been selectively labeled with either a spin label, N-(1-oxyl-2,2,6,6-tetramethyl-4-piperidinyl) iodoacetamide, or with a fluorescent **probe**, S-mercuric-N-dansyl cysteine, presumably at its single cysteine residue (Cys-98) in order to **probe** the interactions of TnC with divalent metals and with other subunits of troponin. The modified protein has the same Ca2+ binding properties as native TnC (Potter, J. D., and Gergely, J. (1975) J. Biol. Chem. 250, 4628), viz. two Ca2+ binding sites at which Mg2+ appears to compete (Ca2+-Mg2+ sites, KCa = 2 X 10(7) M-1) and two sites at which Mg2+ does not compete (Ca2+-specific sites, KCa = 2 X 10(5) M-1). Either Ca2+ or Mg2+ alters the ESR spectrum of spin-labeled TnC in a manner that indicates a decrease in the mobility of the label, Ca2+ having a slightly greater effect. In systems containing both Ca2+ and Mg2+ the mobility of the spin label is identical with that in systems containing Ca2+ alone. The binding constants for Ca2+ and Mg2+ deduced from ESR spectral changes are 10(7) and 10(3) M-1, respectively, and the apparent affinity for Ca2+ decreases by about an order of magnitude on adding 2 mM Mg2+. Thus, the ESR spectral change is associated with binding of Ca2+ to one or both of the Ca2+-Mg2+ sites. Addition of Ca2+ to the **binary** complexes of spin-labeled **TnC** with either troponin T (TnT) or troponin I (**TnI**) produces greater reduction in the mobility of the spin label than in the case of spin-labeled TnC alone, and in the case of the complex with **TnI** the affinity for Ca2+ is increased by an order of magnitude. The fluorescence of dansyl (5-dimethylaminonaphthalene-1-sulfonyl)-labeled TnC is enhanced by Ca2+ binding to both high and low affinity sites with apparent binding constants of 2.6 X 10(7) M-1 and 2.9 X 10(5) M-1, respectively, calculated from the transition midpoints. The presence of 2 mM Mg2+, which produces no effect on dansyl fluorescence itself, in contrast to its effect on the spin label, shifts the high affinity constant to 2 X 10(6) M-1. Spectral changes produced by Ca2+ binding to the TnC-**TnI** complex furnish evidence that the affinity of TnC for Ca2+ is increased in the complex. The reactivity of Cys-98 to the labels and to 5,5'-dithiobis(2-nitrobenzoic acid) (Nbs2) is decreased by Ca2+ or Mg2+ both with native TnC and in 6 M urea. The reaction rate between Cys-98 and Nbs2 decreases to one-half the maximal value at a Ca2+ concentration that suggests binding to the Ca2+-Mg2+ sites. Formation of a **binary** complex between **TnI** and **TnC** reduces the rate of reaction, and there is a further reduction

by Ca2+. The effect of Ca2+ takes place at concentrations that are 1 order
of magnitude lower than in the case of TnC alone. These results suggest that the Ca2+ binding site adjacent to Cys-98 is one of the Ca2+-Mg2+ binding sites.

AB . . . binding component (TnC) of troponin has been selectively labeled with either a spin label, N-(1-oxyl-2,2,6,6-tetramethyl-4-piperidinyl) iodoacetamide, or with a fluorescent **probe**, S-mercuric-N-dansyl cysteine, presumably at its single cysteine residue (Cys-98) in order to **probe** the interactions of TnC with divalent metals and with other subunits of troponin. The modified protein has the same Ca2+. . . change is associated with binding of Ca2+ to one or both of the Ca2+-Mg2+ sites. Addition of Ca2+ to the **binary** complexes of spin-labeled **TnC** with either troponin T (TnT) or troponin I (**TnI**) produces greater reduction in the mobility of the spin label than in the case of spin-labeled TnC alone, and in the case of the complex with **TnI** the affinity for Ca2+ is increased by an order of magnitude. The fluorescence of dansyl (5-dimethylaminonaphthalene-1-sulfonyl)-labeled TnC is enhanced by. . . spin label, shifts the high affinity constant to 2 X 10(6) M-1. Spectral changes produced by Ca2+ binding to the TnC-**TnI** complex furnish evidence that the affinity of TnC for Ca2+ is increased in the complex. The reactivity of Cys-98 to. . . decreases to one-half the maximal value at a Ca2+ concentration that suggests binding to the Ca2+-Mg2+ sites. Formation of a **binary** complex between **TnI** and **TnC** reduces the rate of reaction, and there is a further reduction by Ca2+. The effect of Ca2+ takes place at. . .

L10 ANSWER 3 OF 6 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999132222 MEDLINE
DOCUMENT NUMBER: 99132222 PubMed ID: 9931043
TITLE: Determination of cardiac troponin I forms in the blood of patients with acute myocardial infarction and patients receiving crystalloid or cold blood cardioplegia.
AUTHOR: Giuliani I; Bertinchant J P; Granier C; Laprade M; Chocron S; Toubin G; Etievent J P; Larue C; Trinquier S
CORPORATE SOURCE: Sanofi Diagnostics Pasteur, Z.A. Leopha Rue d'Italie, 69780
Mions, France.
SOURCE: CLINICAL CHEMISTRY, (1999 Feb) 45 (2) 213-22.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990223

AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human **cTnI**. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb. The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and 10 serum samples from patients receiving cold blood cardioplegia during heart surgery were assayed by the three two-site immunoassays. We confirmed that **cTnI** circulates not only in free form but also complexed with the other troponin components (TnC and cTnT). We showed that the predominant form in blood is the **cTnI**-TnC binary complex (IC). Free **cTnI**, the **cTnI**-**cTnT** binary complex, and the **cTnT**-**cTnI**-**TnC ternary** complex were seldom present, and when present, were in small quantities compared with the binary complex IC. Similar results were obtained in both patient populations studied. These observations are essential for the development of new immunoassays with improved clinical sensitivity and for the selection of an appropriate **cTnI** primary calibrator.
AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human **cTnI**. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb. The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and. . . samples from patients receiving cold blood cardioplegia during heart surgery were

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L13 ANSWER 5 OF 19 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1998286707 MEDLINE
DOCUMENT NUMBER: 98286707 PubMed ID: 9625043
TITLE: Characterization of cardiac troponin subunit release into serum after acute myocardial infarction and comparison of assays for troponin T and I. American Association for Clinical Chemistry Subcommittee on cTnI Standardization.
AUTHOR: Wu A H; Feng Y J; Moore R; Apple F S; McPherson P H; Buechler K F; Bodor G
CORPORATE SOURCE: Department of Pathology, Hartford Hospital, CT 06102, USA..
awu@harthosp.org
SOURCE: CLINICAL CHEMISTRY, (1998 Jun) 44 (6 Pt 1) 1198-208.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980625
Last Updated on STN: 19990129
Entered Medline: 19980616

AB We examined the release of cardiac troponin T (cTnT) and I (**cTnI**) into the blood of patients after acute myocardial infarction (AMI). Three postAMI serum samples were applied in separate analytical runs onto a calibrated gel filtration column (Sephacryl S-200), and the proteins were separated by molecular weight. Using commercial cTnT and **cTnI** assays measured on collected fractions, we found that troponin was released into blood as a ternary complex of cTnT-I-C, a binary **complex** of **cTnI**-C, and **free** cTnT, with no **free cTnI** within the limits of the analytical methodologies. The serum samples were also examined after incubation with EDTA and heparin. EDTA broke up troponin complexes into individual subunits, whereas heparin had no effect on the assays tested. We added **free** cTnC subunits to 24 AMI serum samples and found no marked increase in the total **cTnI** concentrations, using an immunoassay that gave higher values for the **cTnI**-C **complex** than **free cTnI**. To characterize the cross-reactivity of cTnT and **cTnI** assays, purified troponin standards in nine different forms were prepared, added to serum and plasma pools, and tested in nine quantitative commercial and pre-market assays for **cTnI** and one approved assay for cTnT. All nine **cTnI** assays recognized each of the troponin I forms (complexed and **free**). In five of these assays, the relative responses for **cTnI** were nearly equimolar. For the remainder, the response was substantially greater for **complexed cTnI** than for **free cTnI**.

Moreover, there was a substantial difference in the absolute concentration of results between **cTnI** assays. The commercial cTnT assay recognized binary and ternary complexes of troponin on a near equimolar basis. We conclude that all assays are useful for detection of cardiac injury. However, there are differences in absolute **cTnI** results due to a lack of mass standardization and heterogeneity in the cross-reactivities of **antibodies** to various troponin I forms.

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L13 ANSWER 9 OF 19 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 97412665 MEDLINE
DOCUMENT NUMBER: 97412665 PubMed ID: 9267317
TITLE: Troponin I is released in bloodstream of patients with acute myocardial infarction not in **free** form but as complex.
AUTHOR: Katrukha A G; Bereznikova A V; Esakova T V; Pettersson K; Lovgren T; Severina M E; Pulkki K; Vuopio-Pulkki L M; Gusev N B
CORPORATE SOURCE: HyTest Ltd., Turku, Finland.
SOURCE: CLINICAL CHEMISTRY, (1997 Aug) 43 (8 Pt 1) 1379-85. Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19970922
Last Updated on STN: 19970922
Entered Medline: 19970911

AB Fourteen monoclonal **antibodies** (mAbs) against human cardiac troponin I (**cTnI**) were generated by commonly used experimental techniques. All these **antibodies**, as well as **antibody** 414 (HyTest), were specific for human **cTnI**. Fifteen **antibodies** thus obtained were tested in a sandwich **cTnI** immunofluorescence assay (altogether 196 combinations). Ten pairs giving the highest sensitivity were selected for further investigation. The effect of **TnI**-TnC **complex** formation on **antibody** interaction with antigen was analyzed. The formation of **TnI**-TnC **complex** results in a significant decrease of the interaction of mAbs with **TnI** for seven of 10 analyzed pairs of **antibodies**. Using two pairs of **cTnI**-specific mAbs, one that recognized only **free cTnI** but not **cTnI** **complexed** with cTnC, and another that could be used for measurement of total **cTnI** (**free cTnI** and **cTnI** in **complex** with cTnC), we demonstrated that the main part of **cTnI** in serum collected from acute myocardial infarction patients is presented in the complex from. We concluded that effective and reliable immunological detection of **TnI** is possible only when **antibodies** used for assay development recognize both **free TnI** and **TnI** **complexed** with other troponin components.

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measurement of total **cTnI** (**free cTnI** and **cTnI** in **complex** with cTnC), we demonstrated that the main part of **cTnI** in serum collected from acute myocardial infarction patients is presented in the complex from. We concluded that effective and reliable immunological detection of **TnI** is possible only when **antibodies** used for assay development recognize both **free TnI** and **TnI complexed** with other troponin components.

L13 ANSWER 15 OF 19 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 86085886 MEDLINE
DOCUMENT NUMBER: 86085886 PubMed ID: 3941095
TITLE: Calcium binding to the low affinity sites in troponin C induces conformational changes in the high affinity domain.
A possible route of information transfer in activation of muscle contraction.
AUTHOR: Grabarek Z; Leavis P C; Gergely J
CONTRACT NUMBER: HL-05811 (NHLBI)
HL-20464 (NHLBI)
HL-5949 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1986 Jan 15) 261 (2) 608-13.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198602
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860214

AB Residues 89-100 of **troponin C** (C89-100) and 96-116 of troponin I (I96-116) interact with each other in the troponin complex (Dalgarno, D.C., Grand, R.J.A., Levine, B.A. Moir, A., J.G., Scott, G.M.M., and Perry, S.V. (1982) FEBS Lett. 150, 54-58) and are necessary for the Ca2+ sensitivity of actomyosin ATPase (Syska, H., Wilkinson, J.M., Grand, R.J.A., and Perry, S.V. (1976) Biochem. J. 153, 375-387 and Grabarek, Z., Drabikowski, W., Leavis, P.C., Rosenfeld, S.S., and Gergely, J. (1981) J. Biol. Chem. 256, 13121-13127). We have studied Ca2+-induced changes in the region C89-100 by monitoring the fluorescence of **troponin C** (TnC) labeled at Cys-98 with 5-(iodoacetamidoethyl)aminonaphthalene-1-sulfonic acid. Equilibrium titration of the labeled TnC with Ca2+ indicates that the **probe** is sensitive to binding to both classes of sites in **free** TnC as well as in its **complex** with **TnI**. When Mg2 X TnC is mixed with Ca2+ in a stopped flow apparatus, there is a rapid fluorescence increase related to Ca2+ binding to the unoccupied sites I and II followed by a slower increase (k = 9.9 s-1) that represents Mg2+-Ca2+ exchange at sites III and IV. In the TnC X **TnI complex**, the fast phase is much larger and the Mg2+-Ca2+ exchange at sites III and IV results in a small decrease rather than an increase in the fluorescence of the **probe**. The possibility is discussed that the fast change in the environment of Cys-98 upon Ca2+ binding to sites I and II may be instrumental in triggering activation of the thin filament by facilitating a contact between C89-100 and I96-116.

AB Residues 89-100 of **troponin C** (C89-100) and 96-116 of troponin I (I96-116) interact with each other in the troponin complex (Dalgarno, D.C., Grand, R.J.A., Levine,. . . (1981) J. Biol. Chem. 256, 13121-13127). We have studied Ca2+-induced changes in the region C89-100 by monitoring the fluorescence of **troponin C** (TnC)

labeled at Cys-98 with 5-(iodoacetamidoethyl)aminonaphthalene-1-sulfonic acid. Equilibrium titration of the labeled TnC with Ca2+ indicates that the **probe** is sensitive to binding to both classes of sites in **free** TnC as well as in its **complex** with **TnI**.
When Mg2 X TnC is mixed with Ca2+ in a stopped flow apparatus, there is a rapid fluorescence increase related. . . a slower increase (k = 9.9 s-1) that represents Mg2+-Ca2+ exchange at sites III and IV. In the TnC X **TnI complex**, the fast phase is much larger and the Mg2+-Ca2+ exchange at sites III and IV results in a small decrease rather than an increase in the fluorescence of the **probe**. The possibility is discussed that the fast change in the environment of Cys-98
upon Ca2+ binding to sites I and. . .

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NEWS 3	Feb 06	Engineering Information Encompass files have new names
NEWS 4	Feb 16	TOXLINE no longer being updated
NEWS 5	Apr 23	Search Derwent WPINDEX by chemical structure
NEWS 6	Apr 23	PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS 7	May 07	DGENE Reload
NEWS 8	Jun 20	Published patent applications (A1) are now in USPATFULL
NEWS 9	JUL 13	New SDI alert frequency now available in Derwent's DWPI and DPCI
NEWS 10	Aug 23	In-process records and more frequent updates now in MEDLINE
NEWS 11	Aug 23	PAGE IMAGES FOR 1947-1966 RECORDS IN CAPLUS AND CA
NEWS 12	Aug 23	Adis Newsletters (ADISNEWS) now available on STN
NEWS 13	Sep 17	IMSworld Pharmaceutical Company Directory name change to PHARMASEARCH

NEWS EXPRESS	August 15 CURRENT WINDOWS VERSION IS V6.0c, CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP), AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
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FILE 'HOME' ENTERED AT 13:32:07 ON 18 SEP 2001

=> file medline,embase,scisearch,biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.15	0.15

FILE 'MEDLINE' ENTERED AT 13:32:26 ON 18 SEP 2001

FILE 'EMBASE' ENTERED AT 13:32:26 ON 18 SEP 2001

FILE 'SCISEARCH' ENTERED AT 13:32:26 ON 18 SEP 2001

FILE 'BIOSIS' ENTERED AT 13:32:26 ON 18 SEP 2001

=> s (troponin c) or ctni or tni

L1 8664 (TROPONIN C) OR CTNI OR TNI

=> s binary (5a) ((troponin c) or ctnc or tnc)

L2 105 BINARY (5A) ((TROPONIN C) OR CTNC OR TNC)

=> s ternary (10a) ((troponin c) or ctnc or tnc) (10a) ((troponin t) or ctnt or tnt)

L3 63 TERNARY (10A) ((TROPONIN C) OR CTNC OR TNC) (10A) ((TROPONIN T)
OR CTNT OR TNT)

=> s l1 (6p) l2

L4 105 L1 (6P) L2

=> s l1 (6p) l3

L5 63 L1 (6P) L3

=> s l1 (6p) (antibod? or receptor? or probe? or antitroponin or anti-troponin)

L6 964 L1 (6P) (ANTIBOD? OR RECEPTOR? OR PROBE? OR ANTITROPONIN OR ANTI-TROPONIN)

=> s l6 and l4

L7 45 L6 AND L4

=> s l6 and l5

L8 20 L6 AND L5

=> dup rem l7

PROCESSING COMPLETED FOR L7
L9 15 DUP REM L7 (30 DUPLICATES REMOVED)

=> dup rem l8

PROCESSING COMPLETED FOR L8
L10 6 DUP REM L8 (14 DUPLICATES REMOVED)

=> d l9 ibib 1-5

L9 ANSWER 1 OF 15 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001361640 MEDLINE
DOCUMENT NUMBER: 21315127 PubMed ID: 11423417
TITLE: Proximity relationships between residue 117 of rabbit skeletal troponin-I and residues in troponin-C and actin.
AUTHOR: Li Z; Gergely J; Tao T
CORPORATE SOURCE: Muscle and Motility Group, Boston Biomedical Research

Institute, Watertown, Massachusetts 02472, USA.
CONTRACT NUMBER: AR21673 (NIAMS)
SOURCE: BIOPHYSICAL JOURNAL, (2001 Jul) 81 (1) 321-33.
Journal code: A5S; 0370626. ISSN: 0006-3495.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200109
ENTRY DATE: Entered STN: 20010917
Last Updated on STN: 20010917
Entered Medline: 20010913

L9 ANSWER 2 OF 15 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001088126 MEDLINE
DOCUMENT NUMBER: 20563843 PubMed ID: 11112516
TITLE: Proximity relationships between residue 6 of troponin I and
residues in troponin C: further evidence for extended conformation of troponin C in the troponin complex.
AUTHOR: Luo Y; Leszyk J; Li B; Gergely J; Tao T
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, Watertown, Massachusetts 02472, USA.
CONTRACT NUMBER: AR21673 (NIAMS)
SOURCE: BIOCHEMISTRY, (2000 Dec 19) 39 (50) 15306-15.
Journal code: A0G. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200101
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010116

L9 ANSWER 3 OF 15 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2001095520 MEDLINE
DOCUMENT NUMBER: 20510320 PubMed ID: 11056032
TITLE: Structural mapping of single cysteine mutants of cardiac troponin I.
AUTHOR: Dong W J; Xing J; Chandra M; Solaro J; Cheung H C
CORPORATE SOURCE: Department of Biochemistry and Molecular Genetics, University of Alabama at Birmingham, 35294-2041, USA.
CONTRACT NUMBER: HL22231 (NHLBI)
HL52508 (NHLBI)
RR10404 (NCRR)
SOURCE: PROTEINS, (2000 Dec 1) 41 (4) 438-47.
Journal code: PTS. ISSN: 0887-3585.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010201

L9 ANSWER 4 OF 15 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999326171 MEDLINE
DOCUMENT NUMBER: 99326171 PubMed ID: 10395953
TITLE: Conformational changes induced in troponin I by interaction
with troponin T and actin/tropomyosin.
AUTHOR: Tao T; Gong B J; Grabarek Z; Gergely J
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, MA 02114, USA.

CONTRACT NUMBER: AR21673 (NIAMS)
RR11301 (NCRR)
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1999 Jul 8) 1450 (3) 423-33.
Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990817

L9 ANSWER 5 OF 15 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1999132222 MEDLINE
DOCUMENT NUMBER: 99132222 PubMed ID: 9931043
TITLE: Determination of cardiac troponin I forms in the blood of patients with acute myocardial infarction and patients receiving crystalloid or cold blood cardioplegia.
AUTHOR: Giuliani I; Bertinchant J P; Granier C; Laprade M; Chocron S; Toubin G; Etievent J P; Larue C; Trinquier S
CORPORATE SOURCE: Sanofi Diagnostics Pasteur, Z.A. Leopha Rue d'Italie, 69780
Mions, France.
SOURCE: CLINICAL CHEMISTRY, (1999 Feb) 45 (2) 213-22.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990223.

=> d l9 kwic 4-5

L9 ANSWER 4 OF 15 MEDLINE DUPLICATE 4
AB Troponin I (**TnI**) is the inhibitory component of the striated muscle Ca2+ regulatory protein troponin (Tn). The other two components of Tn are **troponin C** (TnC), the Ca2+-binding component, and troponin T (TnT), the tropomyosin-binding component. We have used limited chymotryptic digestion to **probe** the local conformation of **TnI** in the free state, the **binary TnC*** **TnI** complex, the ternary **TnC***. **TnI***TnT (Tn) complex, and in the reconstituted Tn*tropomyosin*F-actin filament. The digestion of **TnI** alone or in the TnC***TnI** complex produced initially two major fragments via a cleavage of the peptide bond between Phe100 and Asp101 in the so-called. . . digestion was slower in the absence than in the presence of Ca2+. These results indicate firstly that in both free **TnI** and **TnI** complexed with TnC there is an exposed and flexible site in the inhibitory region. Secondly, TnT affects the conformation of **TnI** in the inhibitory region and also in the region that contains the 140-141 bond. Thirdly, the 140-141 region of **TnI** is likely to interact with actin in the reconstituted thin filament when Ca2+ is absent. These findings are discussed in terms of the role of **TnI** in the mechanism of thin filament regulation, and in light of our previous results [Y. Luo, J.-L. Wu, J. Gergely, T. Tao, Biochemistry 36 (1997) 13449-13454] on the global conformation of **TnI**.

L9 ANSWER 5 OF 15 MEDLINE DUPLICATE 5

AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human **cTnI**. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb. The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and. . . samples from patients receiving cold blood cardioplegia during heart surgery were assayed by the three two-site immunoassays. We confirmed that **cTnI** circulates not only in free form but also complexed with the other troponin components (TnC and cTnT). We showed that the predominant form in blood is the **cTnI**-**TnC binary** complex (IC). Free **cTnI**, the **cTnI**-cTnT **binary** complex, and the cTnT-**cTnI**-**TnC** ternary complex were seldom present, and when present, were in small quantities compared with the binary complex IC. Similar results. . . observations are essential for the development of new immunoassays with improved clinical sensitivity and for the selection of an appropriate **cTnI** primary calibrator.

=> d l9 ibib ab kwic 4-5

L9 ANSWER 4 OF 15 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999326171 MEDLINE
DOCUMENT NUMBER: 99326171 PubMed ID: 10395953
TITLE: Conformational changes induced in troponin I by interaction with troponin T and actin/tropomyosin.
AUTHOR: Tao T; Gong B J; Grabarek Z; Gergely J
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, MA 02114, USA.
CONTRACT NUMBER: AR21673 (NIAMS)
RR11301 (NCRR)
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1999 Jul 8) 1450 (3) 423-33.
Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990817

AB Troponin I (**TnI**) is the inhibitory component of the striated muscle Ca2+ regulatory protein troponin (Tn). The other two components of Tn are **troponin C** (TnC), the Ca2+-binding component, and troponin T (TnT), the tropomyosin-binding component. We have used limited chymotryptic digestion to **probe** the local conformation of **TnI** in the free state, the **binary TnC*** **TnI** complex, the ternary **TnC***. **TnI***TnT (Tn) complex, and in the reconstituted Tn*tropomyosin*F-actin filament. The digestion of **TnI** alone or in the TnC***TnI** complex produced initially two major fragments via a cleavage of the peptide bond between Phe100 and Asp101 in the so-called inhibitory region. In the ternary Tn complex cleavage occurred at a new site between Leu140 and Lys141. In the absence of Ca2+ this was followed by digestion of the

fragment at Leu122 and Met116. In the reconstituted thin filament the same fragments as in the case of the ternary complex were produced, but the rate of digestion was slower in the absence than in the presence of Ca2+. These results indicate firstly that in both free **TnI** and **TnI** complexed with TnC there is an exposed and flexible site in the inhibitory region. Secondly, TnT affects the conformation of **TnI** in the inhibitory region and also in the region that contains the 140-141 bond. Thirdly, the 140-141 region of **TnI** is likely to interact with actin in the reconstituted thin filament when Ca2+ is absent. These findings are discussed in terms of the role of **TnI** in the mechanism of thin filament regulation, and in light of our previous results [Y. Luo, J.-L. Wu, J. Gergely, T. Tao, Biochemistry 36 (1997) 13449-13454] on the global conformation of **TnI**.

AB Troponin I (**TnI**) is the inhibitory component of the striated muscle Ca2+ regulatory protein troponin (Tn). The other two components of Tn are **troponin C** (TnC), the Ca2+-binding component, and troponin T (TnT), the tropomyosin-binding component. We have used limited chymotryptic digestion to **probe** the local conformation of **TnI** in the free state, the **binary TnC*** **TnI** complex, the ternary **TnC***. **TnI***TnT (Tn) complex, and in the reconstituted Tn*tropomyosin*F-actin filament. The digestion of **TnI** alone or in the TnC***TnI** complex produced initially two major fragments via a cleavage of the peptide bond between Phe100 and Asp101 in the so-called. . . digestion was slower in the absence than in the presence of Ca2+. These results indicate firstly that in both free **TnI** and **TnI** complexed with TnC there is an exposed and flexible site in the inhibitory region. Secondly, TnT affects the conformation of **TnI** in the inhibitory region and also in the region that contains the 140-141 bond. Thirdly, the 140-141 region of **TnI** is likely to interact with actin in the reconstituted thin filament when Ca2+ is absent. These findings are discussed in terms of the role of **TnI** in the mechanism of thin filament regulation, and in light of our previous results [Y. Luo, J.-L. Wu, J. Gergely, T. Tao, Biochemistry 36 (1997) 13449-13454] on the global conformation of **TnI**.

L9 ANSWER 5 OF 15 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 1999132222 MEDLINE
DOCUMENT NUMBER: 99132222 PubMed ID: 9931043
TITLE: Determination of cardiac troponin I forms in the blood of patients with acute myocardial infarction and patients receiving crystalloid or cold blood cardioplegia.
AUTHOR: Giuliani I; Bertinchant J P; Granier C; Laprade M; Chocron S; Toubin G; Etievent J P; Larue C; Trinquier S
CORPORATE SOURCE: Sanofi Diagnostics Pasteur, Z.A. Leopha Rue d'Italie, 69780 Mions, France.
SOURCE: CLINICAL CHEMISTRY, (1999 Feb) 45 (2) 213-22. Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990223

AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human

cTnI. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb. The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and 10 serum samples from patients receiving cold blood cardioplegia during heart surgery were assayed by the three two-site immunoassays. We confirmed that **cTnI** circulates not only in free form but also complexed with the other troponin components (TnC and cTnT). We showed that the predominant form in blood is the **cTnI-TnC binary** complex (IC). Free **cTnI**, the **cTnI**-cTnT **binary** complex, and the cTnT-**cTnI-TnC** ternary complex were seldom present, and when present, were in small quantities compared with the binary complex IC. Similar results were obtained in both patient populations studied. These observations are essential for the development of new immunoassays with improved clinical sensitivity and for the selection of an appropriate **cTnI** primary calibrator.

AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human **cTnI**. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb. The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and. . . samples from patients receiving cold blood cardioplegia during heart surgery were assayed by the three two-site immunoassays. We confirmed that **cTnI** circulates not only in free form but also complexed with the other troponin components (TnC and cTnT). We showed that the predominant form in blood is the **cTnI-TnC binary** complex (IC). Free **cTnI**, the **cTnI**-cTnT **binary** complex, and the cTnT-**cTnI-TnC** ternary complex were seldom present, and when present, were in small quantities compared with the binary complex IC. Similar results. . . observations are essential for the development of new immunoassays with improved clinical sensitivity and for the selection of an appropriate **cTnI** primary calibrator.

=> d l9 ibib 6-10

L9 ANSWER 6 OF 15 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 97428233 MEDLINE
DOCUMENT NUMBER: 97428233 PubMed ID: 9283095
TITLE: Troponin T and Ca2+ dependence of the distance between Cys48 and Cys133 of troponin I in the ternary troponin complex and reconstituted thin filaments.
AUTHOR: Luo Y; Wu J L; Gergely J; Tao T
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, Massachusetts 02114, USA.. yinluo@bbri.harvard.edu
CONTRACT NUMBER: R37-AR-21673 (NIAMS)
R37-HL-05949 (NHLBI)
SOURCE: BIOCHEMISTRY, (1997 Sep 9) 36 (36) 11027-35.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971013
Last Updated on STN: 19990129
Entered Medline: 19970930

L9 ANSWER 7 OF 15 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 96215805 MEDLINE
DOCUMENT NUMBER: 96215805 PubMed ID: 8672716
TITLE: A comparison of the properties of the binary and ternary complexes formed by calmodulin and troponin C with two regulatory peptides of phosphorylase kinase.
AUTHOR: Steiner R F; Juminaga D; Albaugh S; Washington H
CORPORATE SOURCE: Department of Chemistry and Biochemistry, University of Maryland, Baltimore 21228, USA.
SOURCE: BIOPHYSICAL CHEMISTRY, (1996 Apr 16) 59 (3) 277-88.
Journal code: A5T; 0403171. ISSN: 0301-4622.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199608
ENTRY DATE: Entered STN: 19960822
Last Updated on STN: 19980206
Entered Medline: 19960813

L9 ANSWER 8 OF 15 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 8
ACCESSION NUMBER: 95066347 EMBASE
DOCUMENT NUMBER: 1995066347
TITLE: Troponin I isoforms and differential effects of acidic pH on soleus and cardiac myofilaments.
AUTHOR: Wattanapermpool J.; Reiser P.J.; Solaro R.J.
CORPORATE SOURCE: Dept. of Physiology and Biophysics, College of Medicine (M/C 901), Univ. of Illinois, 901 South Wolcott,Chicago, IL
60612-7342, United States
SOURCE: American Journal of Physiology - Cell Physiology, (1995) 268/2 37-2 (C323-C330).
ISSN: 0363-6143 CODEN: AJPCDD
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
LANGUAGE: English
SUMMARY LANGUAGE: English

L9 ANSWER 9 OF 15 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 95:132543 SCISEARCH
THE GENUINE ARTICLE: QF550
TITLE: TROPONIN-I ISOFORMS AND DIFFERENTIAL-EFFECTS OF ACIDIC PH ON SOLEUS AND CARDIAC MYOFILAMENTS
AUTHOR: WATTANAPERMPOOL J; REISER P J; SOLARO R J (Reprint)
CORPORATE SOURCE: UNIV ILLINOIS, COLL MED, DEPT PHYSIOL & BIOPHYS, M-C 901, 901 S WOLCOTT, CHICAGO, IL, 60612 (Reprint); UNIV ILLINOIS, COLL MED, DEPT PHYSIOL & BIOPHYS, CHICAGO, IL, 60612
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-CELL PHYSIOLOGY, (FEB 1995)
Vol. 37, No. 2, pp. C323-C330.
ISSN: 0363-6143.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 24
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L9 ANSWER 10 OF 15 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 94375482 MEDLINE
DOCUMENT NUMBER: 94375482 PubMed ID: 8089144
TITLE: NMR studies delineating spatial relationships within the cardiac troponin I-troponin C complex.
AUTHOR: Krudy G A; Kleerekoper Q; Guo X; Howarth J W; Solaro R J; Rosevear P R
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Texas Medical School, Houston 77225.
CONTRACT NUMBER: HL22231 (NHLBI)
HL45724 (NHLBI)
HL49934 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Sep 23) 269 (38) 23731-5.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941031
Last Updated on STN: 19941031
Entered Medline: 19941020

=> d 19 kwic 8,10

L9 ANSWER 8 OF 15 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 8
AB Differences in pH sensitivity of tension generation between developing and
adult cardiac myofilaments, which contain the same isoform of **troponin C** (TnC)(i) have been proposed to be due to troponin I (**TnI**) isoform switching from the slow Skeletal (ss) to cardiac (c) **TnI** isoforms (21). We investigated the effects of acidic pH on Ca2+-activation of force in chemically skinned preparations of adult rat. . . shift in pCa50 in soleus fibers did not change with sarcomere length. Troponin subunit interactions were also investigated, using cardiac **troponin C** (cTnC(IA)) labeled with a fluorescent **probe**, 2-(4'-iodoacetamidoanilino)-naphthalene-6-sulfonic acid. Under acidic conditions, cTnC(IA) demonstrated a decrease in Ca2+affinity. This decrease was amplified both in the **binary** complex **cTnC**(IA)-**cTnI** and in the complex cTnC(IA)-**cTnI**-cTnT-tropomyosin to the same extent. In contrast, substitution of ssTnI for **cTnI** in these complexes produced the same decrease in Ca2+ affinity in response to acidic pH as cTnC(IA) alone.
These results. . . on tension generation and Ca2+ sensitivity between soleus fibers and trabeculae are due to the presence of different isoforms of **TnI**.

L9 ANSWER 10 OF 15 MEDLINE DUPLICATE 9
AB NMR spectroscopy and selective isotope labeling of both recombinant cardiac **troponin C** (cTnC3) and a truncated cardiac troponin I (**cTnI**/NH2) lacking the N-terminal 32-amino acid cardiac-specific sequence have been used to **probe** protein-protein interactions central to muscle contraction. Using [methyl-13C]Met-labeled cTnC3, all 10 cTnC Met residues of Ca(2+)-saturated cTnC3 could be resolved in the two-dimensional heteronuclear single- and multiple-quantum coherence spectrum of the **cTnI**.cTnC complex. Based on the known Met assignments in cTnC3, the largest chemical shift changes were observed for Met81, Met120, Met137,. . . terminus of the central helix. Minimal chemical shift changes were observed for Met45, Met47, and Met103 of cTnC3 in the **cTnI**.cTnC complex. All 6 Met residues in [methyl13C]Met-labeled

cTnI/NH2 could be resolved in the **cTnI**.cTnC complex, suggesting that both **cTnI** and **cTnC** form a stable homogeneous **binary** complex under the conditions of the NMR experiment. In the absence of added protease inhibitors in the **cTnI**.cTnC complex, **cTnI**/NH2 was found to undergo selective proteolysis to yield a 5.5-kDa N-terminal fragment corresponding to residues 33-80. Judging from the NMR spectra of [methyl13C]Met-labeled cTnC3, **cTnI**-(33-80) was sufficient for interaction with the C-terminal domain of cTnC in a manner identical to that observed for native **cTnI**/NH2. However, in the presence of the proteolytic fragment **cTnI**-(33-80), the chemical shift of Met81 was not perturbed from its position in free cTnC3. Thus, residues located C-terminal to Arg80 in **cTnI** appear to be responsible for interaction with the N-terminal half of cTnC. Taken together, these results provide strong evidence for an antiparallel arrangement for the two proteins in the troponin complex such that the N-terminal portion of **cTnI** interacts with the C-terminal domain of cTnC. This interaction likely plays a role in maintaining the stability of the **TnI**.TnC complex.

=> d l9 ibib 11-15

L9 ANSWER 11 OF 15 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 94:17993 SCISEARCH
THE GENUINE ARTICLE: MP293
TITLE: MODULATION OF **TROPONIN-C** BINDING TO TROPONIN-T BY CA2+, **PROBED** BY FLUORESCENCE
AUTHOR: LIN T I (Reprint); MAYADEVI M; DOWBEN R M
CORPORATE SOURCE: NATL TAIWAN UNIV, DEPT CHEM, TAIPEI 106, TAIWAN (Reprint);
BAYLOR UNIV, MED CTR, BAYLOR RES FDN, DALLAS, TX, 75246
COUNTRY OF AUTHOR: TAIWAN; USA
SOURCE: JOURNAL OF THE CHINESE CHEMICAL SOCIETY, (DEC 1993) Vol. 40, No. 6, pp. 607-619.
ISSN: 0009-4536.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: PHYS
LANGUAGE: ENGLISH
REFERENCE COUNT: 34
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L9 ANSWER 12 OF 15 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 91174754 MEDLINE
DOCUMENT NUMBER: 91174754 PubMed ID: 1826079
TITLE: The interaction of troponin C with phosphofructokinase. Comparison with calmodulin.
AUTHOR: Lan J Q; Steiner R F
CORPORATE SOURCE: Department of Chemistry and Biochemistry, University of Maryland Baltimore County 21228.
SOURCE: BIOCHEMICAL JOURNAL, (1991 Mar 1) 274 (Pt 2) 445-51.
Journal code: 9YO; 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199104
ENTRY DATE: Entered STN: 19910512
Last Updated on STN: 19970203
Entered Medline: 19910422

L9 ANSWER 13 OF 15 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 88050893 MEDLINE
DOCUMENT NUMBER: 88050893 PubMed ID: 3676297

TITLE: Interactions of troponin subunits: free energy of binary and ternary complexes.
AUTHOR: Cheung H C; Wang C K; Malik N A
CORPORATE SOURCE: Department of Biochemistry, University of Alabama at Birmingham 35924.
CONTRACT NUMBER: AM25193 (NIADDK)
SOURCE: BIOCHEMISTRY, (1987 Sep 8) 26 (18) 5904-7.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198801
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19880120

L9 ANSWER 14 OF 15 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 87141179 MEDLINE
DOCUMENT NUMBER: 87141179 PubMed ID: 2950237
TITLE: Proximity relationship in the binary complex formed between
troponin I and troponin C.
AUTHOR: Wang C K; Cheung H C
CONTRACT NUMBER: AM25193 (NIADDK)
SOURCE: JOURNAL OF MOLECULAR BIOLOGY, (1986 Oct 5) 191 (3) 509-21.
Journal code: J6V; 2985088R. ISSN: 0022-2836.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198703
ENTRY DATE: Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870330

L9 ANSWER 15 OF 15 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 77071481 MEDLINE
DOCUMENT NUMBER: 77071481 PubMed ID: 187592
TITLE: Effect of Ca2+ binding on troponin C. Changes in spin label
mobility, extrinsic fluorescence, and sulfhydryl reactivity.
AUTHOR: Potter J D; Seidel J C; Leavis P; Lehrer S S; Gergely J
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1976 Dec 10) 251 (23) 7551-6.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197702
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19970203
Entered Medline: 19770216

=> d l9 kwic 11,14,15

L9 ANSWER 11 OF 15 SCISEARCH COPYRIGHT 2001 ISI (R)
TI MODULATION OF **TROPONIN-C** BINDING TO TROPONIN-T BY CA2+, **PROBED** BY FLUORESCENCE
AB The effects of Ca2+ on the binding interaction between **troponin -C** (TnC) and troponin-T (TnT) and between TnC and troponin-I (**TnI**) were studied in both the binary complexes and the presence of

other subunits and tropomyosin (Tm). Rabbit skeletal TnC was. . .
Adding Tm to the system weakened the binding of TnT to TnC by at least 50 - 60 percent. Adding **TnI** to DACM-TnC alone also enhanced the fluorescence (20-22%). An affinity constant 10(8) M-1 for **TnC-TnI binary** complex was obtained. If Tn-I and the labeled TnC were premixed first in 1:1 stoichiometry, then titrated with TnT, a further fluorescence increase (34%) similar to that in the absence of **TnI** was observed. The fit of the binding curve shows that K(a) of TnT to TnC increased (1.5 x 10(6) M-1). When **TnI** was added to the TnC-TnT complex at the end of titration when the fluorescence binding curve became leveled, quenching (12%) occurred. The latter result indicates that **TnI** competes with TnT for the same binding sites on TnC. As binding of TnC is at least 100 times as strong to **TnI** as to TnT, a quenching effect is observed. Furthermore, the conformation of **TnC** in the **TnC-TnI binary** complex may vary from that of TnC alone; binding of TnT to TnC is greatly enhanced (directly or indirectly by **TnI**) in the TnC-**TnI** complex. These results indicate that the variation of binding affinity between TnC and TnT as modulated by Ca2+ may play. . .

L9 ANSWER 14 OF 15 MEDLINE DUPLICATE 12
AB We have determined six molecular distances among four sites in the **binary** complex formed between **troponin C** (**TnC**) and troponin I (**TnI**) by fluorescence resonance energy transfer between donor and acceptor **probes** that were either an intrinsic fluorophore (Trp158 of **TnI**) or extrinsic **probes** attached to the sites. The three extrinsic **probes** were dansylaziridine (DNZ), N'-(iodoacetyl)-N'-(8-sulfo-1-naphthyl)ethylenediamine (IAEDANS) and 5-(iodoacetamido)eosin (IAE). The four fluorophores provided four donor-acceptor pairs: DNZ----IAE, Trp----IAEDANS, IAEDANS----IAE, and Trp----DNZ.. . . from measurements of energy transfer from (1) Met25 (DNZ) to Cys98 (IAE) in TnC, (2) Trp158 to Cys133 (IAEDANS) in **TnI**, (3) Cys98 (IAEDANS) of TnC to Cys133(IAE) of **TnI**, (4) Trp158 of **TnI** to Cys98(IAEDANS) of TnC, and (6) Met25(DNZ) of TnC to Cys133(IAE) of **TnI**. Distance (1) in TnC was little affected when the isolated protein was complexed with **TnI**, whereas distance (2) in **TnI** increased by 6A (29%) when **TnI** was incorporated into the binary complex. In the presence of EGTA, the six donor-acceptor separations (R) in the complex were. . .

L9 ANSWER 15 OF 15 MEDLINE DUPLICATE 13
AB . . . binding component (TnC) of troponin has been selectively labeled with either a spin label, N-(1-oxyl-2,2,6,6-tetramethyl-4-piperidinyl) iodoacetamide, or with a fluorescent **probe**, S-mercuric-N-dansyl cysteine, presumably at its single cysteine residue (Cys-98) in order to **probe** the interactions of TnC with divalent metals and with other subunits of troponin. The modified protein has the same Ca2+. . . change is associated with binding of Ca2+ to one or both of the Ca2+-Mg2+ sites. Addition of Ca2+ to the **binary** complexes of spin-labeled **TnC** with either troponin T (TnT) or troponin I (**TnI**) produces greater reduction in the mobility of the spin label than in the case of spin-labeled TnC alone, and in the case of the complex with **TnI** the affinity for Ca2+ is increased by an order of magnitude. The fluorescence of dansyl (5-dimethylaminonaphthalene-1-sulfonyl)-labeled TnC is enhanced by. . . spin label, shifts the high affinity constant to 2 X 10(6) M-1. Spectral changes produced by Ca2+ binding to the TnC-**TnI** complex furnish evidence that the affinity of TnC for Ca2+ is increased in the complex. The reactivity of Cys-98 to. . . decreases to one-half the maximal value at a Ca2+ concentration that suggests binding to the Ca2+-Mg2+ sites. Formation of a **binary** complex between **TnI** and **TnC** reduces the rate of reaction, and there is a further reduction by Ca2+. The effect of Ca2+ takes place at. . .

=> d l9 ibib ab kwic 15

L9 ANSWER 15 OF 15 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 77071481 MEDLINE
DOCUMENT NUMBER: 77071481 PubMed ID: 187592
TITLE: Effect of Ca2+ binding on troponin C. Changes in spin label
mobility, extrinsic fluorescence, and sulfhydryl reactivity.
AUTHOR: Potter J D; Seidel J C; Leavis P; Lehrer S S; Gergely J
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1976 Dec 10) 251 (23) 7551-6.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197702
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19970203
Entered Medline: 19770216

AB The Ca2+ binding component (TnC) of troponin has been selectively labeled with either a spin label, N-(1-oxyl-2,2,6,6-tetramethyl-4-piperidinyl) iodoacetamide, or with a fluorescent **probe**, S-mercuric-N-dansyl cysteine, presumably at its single cysteine residue (Cys-98) in order to **probe** the interactions of TnC with divalent metals and with other subunits of troponin. The modified protein has the same Ca2+ binding properties as native TnC (Potter, J. D., and Gergely, J. (1975) J. Biol. Chem. 250, 4628), viz. two Ca2+ binding sites at which Mg2+ appears to compete (Ca2+-Mg2+ sites, KCa = 2 X 10(7) M-1) and two sites at which Mg2+ does not compete (Ca2+-specific sites, KCa = 2 X 10(5) M-1). Either Ca2+ or Mg2+ alters the ESR spectrum of spin-labeled TnC in a manner that indicates a decrease in the mobility of the label, Ca2+ having a slightly greater effect. In systems containing both Ca2+ and Mg2+ the mobility of the spin label is identical with that in systems containing Ca2+ alone. The binding constants for Ca2+ and Mg2+ deduced from ESR spectral changes are 10(7) and 10(3) M-1, respectively, and the apparent affinity for Ca2+ decreases by about an order of magnitude on adding 2 mM Mg2+. Thus, the ESR spectral change is associated with binding of Ca2+ to one or both of the Ca2+-Mg2+ sites. Addition of Ca2+ to the **binary** complexes of spin-labeled **TnC** with either troponin T (TnT) or troponin I (**TnI**) produces greater reduction in the mobility of the spin label than in the case of spin-labeled TnC alone, and in the case of the complex with **TnI** the affinity for Ca2+ is increased by an order of magnitude. The fluorescence of dansyl (5-dimethylaminonaphthalene-1-sulfonyl)-labeled TnC is enhanced by Ca2+ binding to both high and low affinity sites with apparent binding constants of 2.6 X 10(7) M-1 and 2.9 X 10(5) M-1, respectively, calculated from the transition midpoints. The presence of 2 mM Mg2+, which produces no effect on dansyl fluorescence itself, in contrast to its effect on the spin label, shifts the high affinity constant to 2 X 10(6) M-1. Spectral changes produced by Ca2+ binding to the TnC-**TnI** complex furnish evidence that the affinity of TnC for Ca2+ is increased in the complex. The reactivity of Cys-98 to the labels and to 5,5'-dithiobis(2-nitrobenzoic acid) (Nbs2) is decreased by Ca2+ or Mg2+ both with native TnC and in 6 M urea. The reaction rate between Cys-98 and Nbs2 decreases to one-half the maximal value at a Ca2+ concentration that suggests binding to the Ca2+-Mg2+ sites. Formation of a **binary** complex between **TnI** and **TnC** reduces the rate of reaction, and there is a further reduction by Ca2+. The effect of Ca2+ takes place at concentrations that are 1 order of magnitude lower than in the case of TnC alone. These results suggest

that the Ca2+ binding site adjacent to Cys-98 is one of the Ca2+-Mg2+ binding sites.

AB . . . binding component (TnC) of troponin has been selectively labeled with either a spin label, N-(1-oxyl-2,2,6,6-tetramethyl-4-piperidinyl) iodoacetamide, or with a fluorescent **probe**, S-mercuric-N-dansyl cysteine, presumably at its single cysteine residue (Cys-98) in order to **probe** the interactions of TnC with divalent metals and with other subunits of troponin. The modified protein has the same Ca2+. . . change is associated with binding of Ca2+ to one or both of the Ca2+-Mg2+ sites. Addition of Ca2+ to the **binary** complexes of spin-labeled **TnC** with either troponin T (TnT) or troponin I (**TnI**) produces greater reduction in the mobility of the spin label than in the case of spin-labeled TnC alone, and in the case of the complex with **TnI** the affinity for Ca2+ is increased by an order of magnitude. The fluorescence of dansyl (5-dimethylaminonaphthalene-1-sulfonyl)-labeled TnC is enhanced by. . . spin label, shifts the high affinity constant to 2 X 10(6) M-1. Spectral changes produced by Ca2+ binding to the TnC-**TnI** complex furnish evidence that the affinity of TnC for Ca2+ is increased in the complex. The reactivity of Cys-98 to. . . decreases to one-half the maximal value at a Ca2+ concentration that suggests binding to the Ca2+-Mg2+ sites. Formation of a **binary** complex between **TnI** and **TnC** reduces the rate of reaction, and there is a further reduction by Ca2+. The effect of Ca2+ takes place at. . .

=> d hi

'HI' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'

The following are valid formats:

The default display format is BIB.

```
ABS ---- AB
ALL ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM,
         ED, AB, CT, ST, RN, CN, NA, GEN
BIB ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
CBIB --- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
DALL --- ALL, delimited for post processing
IABS --- ABS, with a text label
IALL --- ALL, indented with text labels
IBIB --- BIB, indented with text lables
IND ---- CT, ST, RN, CN, NA, GEN
SAM ---- TI, CM, CT, ST, RN, CN, NA, GEN
TRI ---- TI, CM, CT, ST, RN, CN, NA, GEN
TRIAL -- TI, CM, CT, ST, RN, CN, NA, GEN
HIT ---- All fields containing hit terms
HITIND - IND
KWIC --- All hit terms plus 20 words on either side
OCC ---- List of display fields containing hit terms
```

Hit terms will be highlighted in all available fields except CM and PY.

To display a particular field or fields, enter the display field codes. For a list of display field codes, enter 'HELP DFIELDS' at an arrow prompt (=>). Examples of formats include: 'BIB'; 'AB'; 'SO,ST'. You may specify the format fields in any order, and the information will be displayed in the same order as the format specification.

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```
ENTER DISPLAY FORMAT (BIB):his

'HIS' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'

The following are valid formats:

The default display format is BIB.

ABS ---- AB
ALL ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM,
         ED, AB, CT, ST, RN, CN, NA, GEN
BIB ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
CBIB --- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
DALL --- ALL, delimited for post processing
IABS --- ABS, with a text label
IALL --- ALL, indented with text labels
IBIB --- BIB, indented with text lables
IND ---- CT, ST, RN, CN, NA, GEN
SAM ---- TI, CM, CT, ST, RN, CN, NA, GEN
TRI ---- TI, CM, CT, ST, RN, CN, NA, GEN
TRIAL -- TI, CM, CT, ST, RN, CN, NA, GEN
HIT ---- All fields containing hit terms
HITIND - IND
KWIC --- All hit terms plus 20 words on either side
OCC ---- List of display fields containing hit terms

Hit terms will be highlighted in all available fields except CM and
PY.

To display a particular field or fields,  enter the display field
codes.  For a list of display field codes, enter 'HELP DFIELDS'
at an arrow prompt (=>).  Examples of formats include: 'BIB';
'AB'; 'SO,ST'.  You may specify the format fields in any order,
and the information will be displayed in the same order as the
format specification.

The same formats (except for HIT, HITIND, KWIC, and OCC) may be
used with the DISPLAY ACC command to display the record for a
specified Accession Number.
ENTER DISPLAY FORMAT (BIB):1

'1' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'

The following are valid formats:

The default display format is BIB.

ABS ---- AB
ALL ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM,
         ED, AB, CT, ST, RN, CN, NA, GEN
BIB ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
CBIB --- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
DALL --- ALL, delimited for post processing
IABS --- ABS, with a text label
IALL --- ALL, indented with text labels
IBIB --- BIB, indented with text lables
IND ---- CT, ST, RN, CN, NA, GEN
SAM ---- TI, CM, CT, ST, RN, CN, NA, GEN
TRI ---- TI, CM, CT, ST, RN, CN, NA, GEN
TRIAL -- TI, CM, CT, ST, RN, CN, NA, GEN
HIT ---- All fields containing hit terms
HITIND - IND
KWIC --- All hit terms plus 20 words on either side
OCC ---- List of display fields containing hit terms

Hit terms will be highlighted in all available fields except CM and
```

PY.

To display a particular field or fields, enter the display field codes. For a list of display field codes, enter 'HELP DFIELDS' at an arrow prompt (=>). Examples of formats include: 'BIB'; 'AB'; 'SO,ST'. You may specify the format fields in any order, and the information will be displayed in the same order as the format specification.

The same formats (except for HIT, HITIND, KWIC, and OCC) may be used with the DISPLAY ACC command to display the record for a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):ti

L10 ANSWER 1 OF 6 MEDLINE DUPLICATE 1
TI Biochemical and immunological properties of human cardiac troponin I fragments.

=> d his

(FILE 'HOME' ENTERED AT 13:32:07 ON 18 SEP 2001)

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS' ENTERED AT 13:32:26 ON 18 SEP 2001
L1 8664 S (TROPONIN C) OR CTNI OR TNI
L2 105 S BINARY (5A) ((TROPONIN C) OR CTNC OR TNC)
L3 63 S TERNARY (10A) ((TROPONIN C) OR CTNC OR TNC) (10A) ((TROPONIN
L4 105 S L1 (6P) L2
L5 63 S L1 (6P) L3
L6 964 S L1 (6P) (ANTIBOD? OR RECEPTOR? OR PROBE? OR ANTITROPONIN OR A
L7 45 S L6 AND L4
L8 20 S L6 AND L5
L9 15 DUP REM L7 (30 DUPLICATES REMOVED)
L10 6 DUP REM L8 (14 DUPLICATES REMOVED)

=> d l10 ibib 1-6

L10 ANSWER 1 OF 6 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001189932 MEDLINE
DOCUMENT NUMBER: 21175633 PubMed ID: 11277863
TITLE: Biochemical and immunological properties of human cardiac troponin I fragments.
AUTHOR: Morjana N; Clark D; Tal R
CORPORATE SOURCE: Dade Behring Inc., P.O. Box 6101, Glasgow, DE 19714, USA.. morjana@dadebehring.com
SOURCE: BIOTECHNOLOGY AND APPLIED BIOCHEMISTRY, (2001 Apr) 33 (Pt 2) 107-15.
Journal code: AHF; 8609465. ISSN: 0885-4513.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010820
Last Updated on STN: 20010820
Entered Medline: 20010816

L10 ANSWER 2 OF 6 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999326171 MEDLINE
DOCUMENT NUMBER: 99326171 PubMed ID: 10395953

TITLE: Conformational changes induced in troponin I by interaction
with troponin T and actin/tropomyosin.
AUTHOR: Tao T; Gong B J; Grabarek Z; Gergely J
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, MA 02114, USA.
CONTRACT NUMBER: AR21673 (NIAMS)
RR11301 (NCRR)
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1999 Jul 8) 1450 (3) 423-33.
Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990817

L10 ANSWER 3 OF 6 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999132222 MEDLINE
DOCUMENT NUMBER: 99132222 PubMed ID: 9931043
TITLE: Determination of cardiac troponin I forms in the blood of patients with acute myocardial infarction and patients receiving crystalloid or cold blood cardioplegia.
AUTHOR: Giuliani I; Bertinchant J P; Granier C; Laprade M; Chocron S; Toubin G; Etievent J P; Larue C; Trinquier S
CORPORATE SOURCE: Sanofi Diagnostics Pasteur, Z.A. Leopha Rue d'Italie, 69780
Mions, France.
SOURCE: CLINICAL CHEMISTRY, (1999 Feb) 45 (2) 213-22.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990223

L10 ANSWER 4 OF 6 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97428233 MEDLINE
DOCUMENT NUMBER: 97428233 PubMed ID: 9283095
TITLE: Troponin T and Ca2+ dependence of the distance between Cys48 and Cys133 of troponin I in the ternary troponin complex and reconstituted thin filaments.
AUTHOR: Luo Y; Wu J L; Gergely J; Tao T
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, Massachusetts 02114, USA.. yinluo@bbri.harvard.edu
CONTRACT NUMBER: R37-AR-21673 (NIAMS)
R37-HL-05949 (NHLBI)
SOURCE: BIOCHEMISTRY, (1997 Sep 9) 36 (36) 11027-35.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971013
Last Updated on STN: 19990129
Entered Medline: 19970930

L10 ANSWER 5 OF 6 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 88050893 MEDLINE
DOCUMENT NUMBER: 88050893 PubMed ID: 3676297
TITLE: Interactions of troponin subunits: free energy of binary and ternary complexes.
AUTHOR: Cheung H C; Wang C K; Malik N A
CORPORATE SOURCE: Department of Biochemistry, University of Alabama at Birmingham 35924.
CONTRACT NUMBER: AM25193 (NIADDK)
SOURCE: BIOCHEMISTRY, (1987 Sep 8) 26 (18) 5904-7.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198801
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19880120

L10 ANSWER 6 OF 6 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 85023300 MEDLINE
DOCUMENT NUMBER: 85023300 PubMed ID: 6487595
TITLE: Fluorescence lifetime and acrylamide quenching studies of the interactions between troponin subunits.
AUTHOR: Leavis P C; Gowell E; Tao T
CONTRACT NUMBER: AM21673 (NIADDK)
HL20464 (NHLBI)
SOURCE: BIOCHEMISTRY, (1984 Aug 28) 23 (18) 4156-61.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198412
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19841212

=> d l10 ibib ab kwic 2,3,6

L10 ANSWER 2 OF 6 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999326171 MEDLINE
DOCUMENT NUMBER: 99326171 PubMed ID: 10395953
TITLE: Conformational changes induced in troponin I by interaction
with troponin T and actin/tropomyosin.
AUTHOR: Tao T; Gong B J; Grabarek Z; Gergely J
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, MA 02114, USA.
CONTRACT NUMBER: AR21673 (NIAMS)
RR11301 (NCRR)
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1999 Jul 8) 1450 (3) 423-33.
Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990827
Last Updated on STN: 19990827
Entered Medline: 19990817
AB Troponin I (**TnI**) is the inhibitory component of the striated muscle Ca2+ regulatory protein troponin (Tn). The other two components of

Tn are **troponin C** (TnC), the Ca2+-binding component, and troponin T (TnT), the tropomyosin-binding component. We have used limited chymotryptic digestion to **probe** the local conformation of **TnI** in the free state, the binary **TnC*TnI** complex, the **ternary TnC*. TnI*TnT** (Tn) complex, and in the reconstituted Tn*tropomyosin*F-actin filament. The digestion of **TnI** alone or in the TnC*TnI complex produced initially two major fragments via a cleavage of the peptide bond between Phe100 and Asp101 in the so-called inhibitory region. In the ternary Tn complex cleavage occurred at a new site between Leu140 and Lys141. In the absence of Ca2+ this was followed by digestion of the 1-140 fragment at Leu122 and Met116. In the reconstituted thin filament the same fragments as in the case of the ternary complex were produced, but the rate of digestion was slower in the absence than in the presence of Ca2+. These results indicate firstly that in both free **TnI** and **TnI** complexed with TnC there is an exposed and flexible site in the inhibitory region. Secondly, TnT affects the conformation of **TnI** in the inhibitory region and also in the region that contains the 140-141 bond. Thirdly, the 140-141 region of **TnI** is likely to interact with actin in the reconstituted thin filament when Ca2+ is absent. These findings are discussed in terms of the role of **TnI** in the mechanism of thin filament regulation, and in light of our previous results [Y. Luo, J.-L. Wu, J. Gergely, T. Tao, Biochemistry 36 (1997) 13449-13454] on the global conformation of **TnI**.

AB Troponin I (**TnI**) is the inhibitory component of the striated muscle Ca2+ regulatory protein troponin (Tn). The other two components of Tn are **troponin C** (TnC), the Ca2+-binding component, and troponin T (TnT), the tropomyosin-binding component. We have used limited chymotryptic digestion to **probe** the local conformation of **TnI** in the free state, the binary **TnC*TnI** complex, the **ternary TnC*. TnI*TnT** (Tn) complex, and in the reconstituted Tn*tropomyosin*F-actin filament. The digestion of **TnI** alone or in the TnC*TnI complex produced initially two major fragments via a cleavage of the peptide bond between Phe100 and Asp101 in the so-called. . . digestion was slower in the absence than in the presence of Ca2+. These results indicate firstly that in both free **TnI** and **TnI** complexed with TnC there is an exposed and flexible site in the inhibitory region. Secondly, TnT affects the conformation of **TnI** in the inhibitory region and also in the region that contains the 140-141 bond. Thirdly, the 140-141 region of **TnI** is likely to interact with actin in the reconstituted thin filament when Ca2+ is absent. These findings are discussed in terms of the role of **TnI** in the mechanism of thin filament regulation, and in light of our previous results [Y. Luo, J.-L. Wu, J. Gergely, T. Tao, Biochemistry 36 (1997) 13449-13454] on the global conformation of **TnI**.

L10 ANSWER 3 OF 6 MEDLINE DUPLICATE 3

ACCESSION NUMBER: 1999132222 MEDLINE
DOCUMENT NUMBER: 99132222 PubMed ID: 9931043
TITLE: Determination of cardiac troponin I forms in the blood of patients with acute myocardial infarction and patients receiving crystalloid or cold blood cardioplegia.
AUTHOR: Giuliani I; Bertinchant J P; Granier C; Laprade M; Chocron S; Toubin G; Etievent J P; Larue C; Trinquier S
CORPORATE SOURCE: Sanofi Diagnostics Pasteur, Z.A. Leopha Rue d'Italie, 69780 Mions, France.
SOURCE: CLINICAL CHEMISTRY, (1999 Feb) 45 (2) 213-22.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990223

AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human **cTnI**. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb. The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and 10 serum samples from patients receiving cold blood cardioplegia during heart surgery were assayed by the three two-site immunoassays. We confirmed that **cTnI** circulates not only in free form but also complexed with the other troponin components (TnC and cTnT). We showed that the predominant form in blood is the **cTnI**-TnC binary complex (IC). Free **cTnI**, the **cTnI-cTnT** binary complex, and the **cTnT-cTnI-TnC ternary** complex were seldom present, and when present, were in small quantities compared with the binary complex IC. Similar results were obtained in both patient populations studied. These observations are essential for the development of new immunoassays with improved clinical sensitivity and for the selection of an appropriate **cTnI** primary calibrator.

AB To determine the forms of cardiac troponin I (**cTnI**) circulating in the bloodstream of patients with acute myocardial infarction (AMI) and patients receiving a cardioplegia during heart surgery, we developed three immunoenzymatic sandwich assays. The first assay involves the combination of two monoclonal **antibodies** (mAbs) specific for human **cTnI**. The second assay involves the combination of a mAb specific for **troponin C** (TnC) and an anti-**cTnI** mAb. The third assay was a combination of a mAb specific for human cardiac troponin T (cTnT) and an anti-**cTnI** mAb. Fifteen serum samples from patients with AMI, 10 serum samples from patients receiving crystalloid cardioplegia during heart surgery, and. . . samples from patients receiving cold blood cardioplegia during heart surgery were assayed by the three two-site immunoassays. We confirmed that **cTnI** circulates not only in free form but also complexed with the other troponin components (TnC and cTnT). We showed that the predominant form in blood is the **cTnI**-TnC binary complex (IC). Free **cTnI**, the **cTnI-cTnT** binary complex, and the **cTnT-cTnI-TnC ternary** complex were seldom present, and when present, were in small quantities compared with the binary complex IC. Similar results were. . . observations are essential for the development of new immunoassays with improved clinical sensitivity and for the selection of an appropriate **cTnI** primary calibrator.

L10 ANSWER 6 OF 6 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 85023300 MEDLINE
DOCUMENT NUMBER: 85023300 PubMed ID: 6487595
TITLE: Fluorescence lifetime and acrylamide quenching studies of the interactions between troponin subunits.
AUTHOR: Leavis P C; Gowell E; Tao T
CONTRACT NUMBER: AM21673 (NIADDK)
HL20464 (NHLBI)
SOURCE: BIOCHEMISTRY, (1984 Aug 28) 23 (18) 4156-61.

Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198412
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19841212

AB Fluorescence lifetime and acrylamide quenching studies were carried out to characterize the interactions between the subunits of troponin under various conditions of metal ion binding. **Troponin C** was labeled at Cys-98 with N-(iodoacetyl)-N'-(5-sulfo-1-naphthyl)ethylenediamine. In the presence of Ca2+, the fluorescence decay of labeled **troponin C** (TnC*) was monoexponential, lifetime tau = 15.5 ns and quenching rate constant kq = 2.97 X 10(8) M-1 s-1. In the absence of Ca2+, the decay was resolvable into a major component with tau = 11.9 ns and a minor component with tau = 20.5 ns, with corresponding values of kq = 4.80 X 10(8) and 0.66 X 10(8) M-1 s-1, respectively. Upon the binding of either troponin I (**TnI**) or troponin T (TnT) in the presence of Ca2+, tau increased to approximately 18 ns, and kq decreased to approximately 0.8 X 10(8) M-1 s-1. For the Ca2+ form of the **TnC*-TnI-TnT ternary** complex, values of tau = 17.6 ns and kq = 1.73 X 10(8) M-1 s-1 were obtained. These values did not vary significantly when Ca2+ was removed, or when Mg2+ replaced Ca2+. These findings were interpreted as follows: the region around Cys-98 of TnC* adopts a looser conformation upon the removal of Ca2+ from the high-affinity sites. Both **TnI** and TnT bind to TnC* in the region containing Cys-98. The **probe** is shielded from the solvent to a greater extent in the binary complexes than in the ternary complex.(ABSTRACT TRUNCATED AT 250 WORDS)

AB . . . studies were carried out to characterize the interactions between the subunits of troponin under various conditions of metal ion binding. **Troponin C** was labeled at Cys-98 with N-(iodoacetyl)-N'-(5-sulfo-1-naphthyl)ethylenediamine. In the presence of Ca2+, the fluorescence decay of labeled **troponin C** (TnC*) was monoexponential, lifetime tau = 15.5 ns and quenching rate constant kq = 2.97 X 10(8) M-1 s-1. In. . . of kq = 4.80 X 10(8) and 0.66 X 10(8) M-1 s-1, respectively. Upon the binding of either troponin I (**TnI**) or troponin T (TnT) in the presence of Ca2+, tau increased to approximately 18 ns, and kq decreased to approximately 0.8 X 10(8) M-1 s-1. For the Ca2+ form of the **TnC*-TnI-TnT ternary** complex, values of tau = 17.6 ns and kq = 1.73 X 10(8) M-1 s-1 were obtained. These values did. . . the region around Cys-98 of TnC* adopts a looser conformation upon the removal of Ca2+ from the high-affinity sites. Both **TnI** and TnT bind to TnC* in the region containing Cys-98. The **probe** is shielded from the solvent to a greater extent in the binary complexes than in the ternary complex.(ABSTRACT TRUNCATED AT. . .

=> d his

(FILE 'HOME' ENTERED AT 13:32:07 ON 18 SEP 2001)

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS' ENTERED AT 13:32:26 ON 18 SEP 2001

L1 8664 S (TROPONIN C) OR CTNI OR TNI
L2 105 S BINARY (5A) ((TROPONIN C) OR CTNC OR TNC)
L3 63 S TERNARY (10A) ((TROPONIN C) OR CTNC OR TNC) (10A) ((TROPONIN

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L4          105 S L1 (6P) L2
L5           63 S L1 (6P) L3
L6          964 S L1 (6P) (ANTIBOD? OR RECEPTOR? OR PROBE? OR ANTITROPONIN OR
A
L7           45 S L6 AND L4
L8           20 S L6 AND L5
L9           15 DUP REM L7 (30 DUPLICATES REMOVED)
L10           6 DUP REM L8 (14 DUPLICATES REMOVED)

=> s l1 (6a) (bound or complex?) and (unbound or free or uncomplex?)

L11        189 L1 (6A) (BOUND OR COMPLEX?) AND (UNBOUND OR FREE OR
UNCOMPLEX?)

=> s l11 and l6

L12         61 L11 AND L6

=> dup rem l12

PROCESSING COMPLETED FOR L12
L13          19 DUP REM L12 (42 DUPLICATES REMOVED)

=> d l13 ibib 1-5


L13  ANSWER 1 OF 19     MEDLINE                          DUPLICATE 1
ACCESSION NUMBER:   2001189932     MEDLINE
DOCUMENT NUMBER:    21175633   PubMed ID: 11277863
TITLE:              Biochemical and immunological properties of human cardiac
                    troponin I fragments.
AUTHOR:             Morjana N; Clark D; Tal R
CORPORATE SOURCE:   Dade Behring Inc., P.O. Box 6101, Glasgow, DE 19714, USA..
                    morjana@dadebehring.com
SOURCE:             BIOTECHNOLOGY AND APPLIED BIOCHEMISTRY, (2001 Apr) 33 (Pt
                    2) 107-15.
                    Journal code: AHF; 8609465. ISSN: 0885-4513.
PUB. COUNTRY:       England: United Kingdom
                    Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200108
ENTRY DATE:         Entered STN: 20010820
                    Last Updated on STN: 20010820
                    Entered Medline: 20010816

L13  ANSWER 2 OF 19     MEDLINE                          DUPLICATE 2
ACCESSION NUMBER:   1999326171     MEDLINE
DOCUMENT NUMBER:    99326171   PubMed ID: 10395953
TITLE:              Conformational changes induced in troponin I by
interaction
                    with troponin T and actin/tropomyosin.
AUTHOR:             Tao T; Gong B J; Grabarek Z; Gergely J
CORPORATE SOURCE:   Muscle Research Group, Boston Biomedical Research
                    Institute, 20 Staniford Street, Boston, MA 02114, USA.
CONTRACT NUMBER:    AR21673 (NIAMS)
                    RR11301 (NCRR)
SOURCE:             BIOCHIMICA ET BIOPHYSICA ACTA, (1999 Jul 8) 1450 (3)
                    423-33.
                    Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY:       Netherlands
                    Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        199908
ENTRY DATE:         Entered STN: 19990827
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Last Updated on STN: 19990827
Entered Medline: 19990817

L13 ANSWER 3 OF 19 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999132222 MEDLINE
DOCUMENT NUMBER: 99132222 PubMed ID: 9931043
TITLE: Determination of cardiac troponin I forms in the blood of patients with acute myocardial infarction and patients receiving crystalloid or cold blood cardioplegia.
AUTHOR: Giuliani I; Bertinchant J P; Granier C; Laprade M; Chocron S; Toubin G; Etievent J P; Larue C; Trinquier S
CORPORATE SOURCE: Sanofi Diagnostics Pasteur, Z.A. Leopha Rue d'Italie, 69780
Mions, France.
SOURCE: CLINICAL CHEMISTRY, (1999 Feb) 45 (2) 213-22.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990223

L13 ANSWER 4 OF 19 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1998226730 MEDLINE
DOCUMENT NUMBER: 98226730 PubMed ID: 9560191
TITLE: Crystal structure of **troponin C** in **complex** with troponin I fragment at 2.3-A resolution.
AUTHOR: Vassylyev D G; Takeda S; Wakatsuki S; Maeda K; Maeda Y
CORPORATE SOURCE: International Institute for Advanced Research, Central Research Laboratories, Matsushita Electric Industrial Co., Ltd., 3-4 Hikaridai, Seika, Kyoto, 619-02, Japan.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1998 Apr 28) 95 (9) 4847-52.
Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: PDB-1A2X
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980611
Last Updated on STN: 19980611
Entered Medline: 19980604

L13 ANSWER 5 OF 19 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1998286707 MEDLINE
DOCUMENT NUMBER: 98286707 PubMed ID: 9625043
TITLE: Characterization of cardiac troponin subunit release into serum after acute myocardial infarction and comparison of assays for troponin T and I. American Association for Clinical Chemistry Subcommittee on cTnI Standardization.
AUTHOR: Wu A H; Feng Y J; Moore R; Apple F S; McPherson P H; Buechler K F; Bodor G
CORPORATE SOURCE: Department of Pathology, Hartford Hospital, CT 06102, USA..
awu@harthosp.org
SOURCE: CLINICAL CHEMISTRY, (1998 Jun) 44 (6 Pt 1) 1198-208.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980625
Last Updated on STN: 19990129
Entered Medline: 19980616

=> d l13 ibib ab kwic 5

L13 ANSWER 5 OF 19 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 1998286707 MEDLINE
DOCUMENT NUMBER: 98286707 PubMed ID: 9625043
TITLE: Characterization of cardiac troponin subunit release into serum after acute myocardial infarction and comparison of assays for troponin T and I. American Association for Clinical Chemistry Subcommittee on cTnI Standardization.
AUTHOR: Wu A H; Feng Y J; Moore R; Apple F S; McPherson P H; Buechler K F; Bodor G
CORPORATE SOURCE: Department of Pathology, Hartford Hospital, CT 06102, USA..
awu@harthosp.org
SOURCE: CLINICAL CHEMISTRY, (1998 Jun) 44 (6 Pt 1) 1198-208.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980625
Last Updated on STN: 19990129
Entered Medline: 19980616

AB We examined the release of cardiac troponin T (cTnT) and I (**cTnI**) into the blood of patients after acute myocardial infarction (AMI). Three postAMI serum samples were applied in separate analytical runs onto a calibrated gel filtration column (Sephacryl S-200), and the proteins were separated by molecular weight. Using commercial cTnT and **cTnI** assays measured on collected fractions, we found that troponin was released into blood as a ternary complex of cTnT-I-C, a binary **complex** of **cTnI**-C, and **free** cTnT, with no **free cTnI** within the limits of the analytical methodologies. The serum samples were also examined after incubation with EDTA and heparin. EDTA broke up troponin complexes into individual subunits, whereas heparin had no effect on the assays tested. We added **free** cTnC subunits to 24 AMI serum samples and found no marked increase in the total **cTnI** concentrations, using an immunoassay that gave higher values for the **cTnI**-C **complex** than **free cTnI**. To characterize the cross-reactivity of cTnT and **cTnI** assays, purified troponin standards in nine different forms were prepared, added to serum and plasma pools, and tested in nine quantitative commercial and pre-market assays for **cTnI** and one approved assay for cTnT. All nine **cTnI** assays recognized each of the troponin I forms (complexed and **free**). In five of these assays, the relative responses for **cTnI** were nearly equimolar. For the remainder, the response was substantially greater for **complexed cTnI** than for **free cTnI**.
Moreover, there was a substantial difference in the absolute concentration of results between **cTnI** assays. The commercial cTnT assay recognized binary and ternary complexes of troponin on a near equimolar basis. We conclude that all assays are useful for detection of cardiac injury. However, there are differences in absolute **cTnI** results due to a lack of mass standardization and heterogeneity in the cross-reactivities of **antibodies** to various troponin I forms.
AB We examined the release of cardiac troponin T (cTnT) and I (**cTnI**) into the blood of patients after acute myocardial infarction (AMI). Three postAMI serum samples were applied in separate analytical runs.

. onto a calibrated gel filtration column (Sephacryl S-200), and the proteins were separated by molecular weight. Using commercial cTnT and **cTnI** assays measured on collected fractions, we found that troponin was released into blood as a ternary complex of cTnT-I-C, a binary **complex** of **cTnI**-C, and **free** cTnT, with no **free cTnI** within the limits of the analytical methodologies. The serum samples were also examined after incubation with EDTA and heparin. EDTA broke up troponin complexes into individual subunits, whereas heparin had no effect on the assays tested. We added **free** cTnC subunits to 24 AMI serum samples and found no marked increase in the total **cTnI** concentrations, using an immunoassay that gave higher values for the **cTnI**-C **complex** than **free cTnI**. To characterize the cross-reactivity of cTnT and **cTnI** assays, purified troponin standards in nine different forms were prepared, added to serum and plasma pools, and tested in nine quantitative commercial and pre-market assays for **cTnI** and one approved assay for cTnT. All nine **cTnI** assays recognized each of the troponin I forms (complexed and **free**). In five of these assays, the relative responses for **cTnI** were nearly equimolar. For the remainder, the response was substantially greater for **complexed cTnI** than for **free cTnI**. Moreover, there was a substantial difference in the absolute concentration of results between **cTnI** assays. The commercial cTnT assay recognized binary and ternary complexes of troponin on a near equimolar basis. We conclude that all assays are useful for detection of cardiac injury. However, there are differences in absolute **cTnI** results due to a lack of mass standardization and heterogeneity in the cross-reactivities of **antibodies** to various troponin I forms.

=> d l13 ibib 6-10

L13 ANSWER 6 OF 19 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 1999018184 MEDLINE
DOCUMENT NUMBER: 99018184 PubMed ID: 9799527
TITLE: Real-time analysis of immunogen complex reaction kinetics using surface plasmon resonance.
AUTHOR: Yu Y Y; Van Wie B J; Koch A R; Moffett D F; Davis W C
CORPORATE SOURCE: Department of Chemical Engineering, Washington State University, Pullman, Washington 99164, USA.
SOURCE: ANALYTICAL BIOCHEMISTRY, (1998 Oct 15) 263 (2) 158-68. Journal code: 4NK; 0370535. ISSN: 0003-2697.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981216

L13 ANSWER 7 OF 19 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 1999106500 MEDLINE
DOCUMENT NUMBER: 99106500 PubMed ID: 9889826
TITLE: The crystal structure of **troponin C** in **complex** with N-terminal fragment of troponin I. The mechanism of how the inhibitory action of troponin I is released by Ca(2+)-binding to troponin C.
AUTHOR: Vassylyev D G; Takeda S; Wakatsuki S; Maeda K; Maeda Y
CORPORATE SOURCE: International Institute for Advanced Research, Central Research Laboratories, Matsushita Electric Industrial Co., Ltd., Kyoto, Japan.
SOURCE: ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1998) 453 157-67.

Journal code: 2LU; 0121103. ISSN: 0065-2598.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990210

L13 ANSWER 8 OF 19 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 97428233 MEDLINE
DOCUMENT NUMBER: 97428233 PubMed ID: 9283095
TITLE: Troponin T and Ca2+ dependence of the distance between Cys48 and Cys133 of troponin I in the ternary troponin complex and reconstituted thin filaments.
AUTHOR: Luo Y; Wu J L; Gergely J; Tao T
CORPORATE SOURCE: Muscle Research Group, Boston Biomedical Research Institute, 20 Staniford Street, Boston, Massachusetts 02114, USA.. yinluo@bbri.harvard.edu
CONTRACT NUMBER: R37-AR-21673 (NIAMS)
R37-HL-05949 (NHLBI)
SOURCE: BIOCHEMISTRY, (1997 Sep 9) 36 (36) 11027-35.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971013
Last Updated on STN: 19990129
Entered Medline: 19970930

L13 ANSWER 9 OF 19 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 97412665 MEDLINE
DOCUMENT NUMBER: 97412665 PubMed ID: 9267317
TITLE: Troponin I is released in bloodstream of patients with acute myocardial infarction not in **free** form but as complex.
AUTHOR: Katrukha A G; Bereznikova A V; Esakova T V; Pettersson K; Lovgren T; Severina M E; Pulkki K; Vuopio-Pulkki L M; Gusev N B
CORPORATE SOURCE: HyTest Ltd., Turku, Finland.
SOURCE: CLINICAL CHEMISTRY, (1997 Aug) 43 (8 Pt 1) 1379-85.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19970922
Last Updated on STN: 19970922
Entered Medline: 19970911

L13 ANSWER 10 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:334335 BIOSIS
DOCUMENT NUMBER: PREV199799633538
TITLE: Kinetics of liberation of **free** and total cardiac troponin I (cTnI) in serums of patients with acute myocardial infarction (AMI.
AUTHOR(S): Katrukha, A. (1); Beresnikova, A.; Petterson, K.; Lovgren, T.; Gusev, N.; Esakova, T.; Pulkki, K.; Vuopio-Pulkki, L. M.
CORPORATE SOURCE: (1) Hydest LTD., Turku Finland
SOURCE: Clinical Chemistry, (1997) Vol. 43, No. 6 PART 2, pp. S116.

Meeting Info.: 49th Annual Meeting of the American Association for Clinical Chemistry Atlanta, Georgia, USA July 20-24, 1997
ISSN: 0009-9147.
DOCUMENT TYPE: Conference; Abstract; Conference
LANGUAGE: English

=> d l13 ibib ab kwic 7,9,10

L13 ANSWER 7 OF 19 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 1999106500 MEDLINE
DOCUMENT NUMBER: 99106500 PubMed ID: 9889826
TITLE: The crystal structure of **troponin C** in **complex** with N-terminal fragment of troponin I. The mechanism of how the inhibitory action of troponin I is released by Ca(2+)-binding to troponin C.
AUTHOR: Vassylyev D G; Takeda S; Wakatsuki S; Maeda K; Maeda Y
CORPORATE SOURCE: International Institute for Advanced Research, Central Research Laboratories, Matsushita Electric Industrial Co., Ltd., Kyoto, Japan.
SOURCE: ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1998) 453 157-67.
Journal code: 2LU; 0121103. ISSN: 0065-2598.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990210

AB Troponin (Tn), the **complex** of three subunits (TnC, **TnI**, and TnT), plays a key role in Ca2+ dependent regulation of muscle contraction. To elucidate the interactions between the Tn subunits and the conformation of TnC in the Tn complex, we have determined the crystal structure of TnC in **complex** with the N-terminal fragment of **TnI** (TnI1-47). The structure was solved by single isomorphous replacement method in combination with multiple wavelength anomalous dispersion data. The refinement converged to a crystallographic R-factor of 22.2% (R-**free** = 32.6%). The central, connecting alpha-helix observed in the structure of **uncomplexed** TnC (TnCfree) is unwound at the center and bent by 90 degrees. As a result, the TnC in the complex has a compact globular shape with direct interactions between the N- and C-lobes, in contrast to the elongated dumb-bell shaped molecule of **uncomplexed** TnC. The 31-residue long TnI1-47 alpha-helix stretches on the surface of TnC and stabilizes its compact conformation by multiple contacts with both TnC lobes. The amphiphilic C-terminal end of the TnI1-47 alpha-helix is tightly bound in the hydrophobic pocket of the TnC C-lobe through 38 van der Waals interactions. The results indicate the major difference between integrated (TnC) and isolated (calmodulin) Ca2+ **receptors**. The TnC/TnI1-47 structure suggests the model for a novel regulatory **TnI** segment **bound** to TnC and implies the mechanism of how Tn regulates the muscle contraction.

TI The crystal structure of **troponin C** in **complex** with N-terminal fragment of troponin I. The mechanism of how the inhibitory action of troponin I is released by Ca(2+)-binding. . .

AB Troponin (Tn), the **complex** of three subunits (TnC, **TnI**, and TnT), plays a key role in Ca2+ dependent regulation of muscle contraction. To elucidate the interactions between the Tn subunits and the conformation of TnC in the Tn complex, we have determined the crystal structure of TnC in **complex** with the N-terminal fragment of **TnI** (TnI1-47). The structure was solved by single isomorphous

replacement method in combination with multiple wavelength anomalous dispersion data. The refinement converged to a crystallographic R-factor of 22.2% (R-**free** = 32.6%). The central, connecting alpha-helix observed in the structure of **uncomplexed** TnC (TnCfree) is unwound at the center and bent by 90 degrees. As a result, the TnC in the complex. . . compact globular shape with direct interactions between the N- and C-lobes, in contrast to the elongated dumb-bell shaped molecule of **uncomplexed** TnC. The 31-residue long TnI1-47 alpha-helix stretches on the surface of TnC and stabilizes its compact conformation by multiple contacts. . . C-lobe through 38 van der Waals interactions. The results indicate the major difference between integrated (TnC) and isolated (calmodulin) Ca2+ **receptors**. The TnC/TnI1-47 structure suggests the model for a novel regulatory **TnI** segment **bound** to TnC and implies the mechanism of how Tn regulates the muscle contraction.

L13 ANSWER 9 OF 19 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 97412665 MEDLINE
DOCUMENT NUMBER: 97412665 PubMed ID: 9267317
TITLE: Troponin I is released in bloodstream of patients with acute myocardial infarction not in **free** form but as complex.
AUTHOR: Katrukha A G; Bereznikova A V; Esakova T V; Pettersson K; Lovgren T; Severina M E; Pulkki K; Vuopio-Pulkki L M; Gusev N B
CORPORATE SOURCE: HyTest Ltd., Turku, Finland.
SOURCE: CLINICAL CHEMISTRY, (1997 Aug) 43 (8 Pt 1) 1379-85.
Journal code: DBZ; 9421549. ISSN: 0009-9147.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19970922
Last Updated on STN: 19970922
Entered Medline: 19970911

AB Fourteen monoclonal **antibodies** (mAbs) against human cardiac troponin I (**cTnI**) were generated by commonly used experimental techniques. All these **antibodies**, as well as **antibody** 414 (HyTest), were specific for human **cTnI**. Fifteen **antibodies** thus obtained were tested in a sandwich **cTnI** immunofluorescence assay (altogether 196 combinations). Ten pairs giving the highest sensitivity were selected for further investigation. The effect of **TnI**-TnC **complex** formation on **antibody** interaction with antigen was analyzed. The formation of **TnI**-TnC **complex** results in a significant decrease of the interaction of mAbs with **TnI** for seven of 10 analyzed pairs of **antibodies**. Using two pairs of **cTnI**-specific mAbs, one that recognized only **free cTnI** but not **cTnI** **complexed** with cTnC, and another that could be used for measurement of total **cTnI** (**free cTnI** and **cTnI** in **complex** with cTnC), we demonstrated that the main part of **cTnI** in serum collected from acute myocardial infarction patients is presented in the complex from. We concluded that effective and reliable immunological detection of **TnI** is possible only when **antibodies** used for assay development recognize both **free TnI** and **TnI** **complexed** with other troponin components.

TI Troponin I is released in bloodstream of patients with acute myocardial infarction not in **free** form but as complex.

AB Fourteen monoclonal **antibodies** (mAbs) against human cardiac troponin I (**cTnI**) were generated by commonly used experimental techniques. All these **antibodies**, as well as **antibody**

414 (HyTest), were specific for human **cTnI**. Fifteen **antibodies** thus obtained were tested in a sandwich **cTnI** immunofluorescence assay (altogether 196 combinations). Ten pairs giving the highest sensitivity were selected for further investigation. The effect of **TnI**-TnC **complex** formation on **antibody** interaction with antigen was analyzed. The formation of **TnI**-TnC **complex** results in a significant decrease of the interaction of mAbs with **TnI** for seven of 10 analyzed pairs of **antibodies**. Using two pairs of **cTnI**-specific mAbs, one that recognized only **free cTnI** but not **cTnI complexed** with cTnC, and another that could be used for measurement of total **cTnI** (**free cTnI** and **cTnI** in **complex** with cTnC), we demonstrated that the main part of **cTnI** in serum collected from acute myocardial infarction patients is presented in the complex from. We concluded that effective and reliable immunological detection of **TnI** is possible only when **antibodies** used for assay development recognize both **free TnI** and **TnI complexed** with other troponin components.

L13 ANSWER 10 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:334335 BIOSIS
DOCUMENT NUMBER: PREV199799633538
TITLE: Kinetics of liberation of **free** and total cardiac troponin I (cTnI) in serums of patients with acute myocardial infarction (AMI.
AUTHOR(S): Katrukha, A. (1); Beresnikova, A.; Petterson, K.; Lovgren, T.; Gusev, N.; Esakova, T.; Pulkki, K.; Vuopio-Pulkki, L. M.
CORPORATE SOURCE: (1) Hydest LTD., Turku Finland
SOURCE: Clinical Chemistry, (1997) Vol. 43, No. 6 PART 2, pp. S116.
Meeting Info.: 49th Annual Meeting of the American Association for Clinical Chemistry Atlanta, Georgia, USA July 20-24, 1997
ISSN: 0009-9147.
DOCUMENT TYPE: Conference; Abstract; Conference
LANGUAGE: English
TI Kinetics of liberation of **free** and total cardiac troponin I (cTnI) in serums of patients with acute myocardial infarction (AMI.
IT Miscellaneous Descriptors
ACUTE MYOCARDIAL INFARCTION; ANALYTICAL METHOD; CARDIAC TROPONIN I; CARDIOVASCULAR MEDICINE; CHEST PAIN; CLINICAL CHEMISTRY; **FREE** LEVEL; HEART DISEASE; IMMUNOFLUORESCENCE DETECTION; LIBERATION KINETICS; METHODOLOGY; MONOCLONAL **ANTIBODIES**; PATIENT; SERUM; TOTAL LEVEL; TROPONIN I-**TROPONIN C COMPLEX** ; VASCULAR DISEASE

=> d l13 ibib 11-15

L13 ANSWER 11 OF 19 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 94375482 MEDLINE
DOCUMENT NUMBER: 94375482 PubMed ID: 8089144
TITLE: NMR studies delineating spatial relationships within the cardiac troponin I-**troponin C complex**.
AUTHOR: Krudy G A; Kleerekoper Q; Guo X; Howarth J W; Solaro R J; Rosevear P R
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Texas Medical School, Houston 77225.
CONTRACT NUMBER: HL22231 (NHLBI)
HL45724 (NHLBI)
HL49934 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Sep 23) 269 (38)

23731-5.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941031
Last Updated on STN: 19941031
Entered Medline: 19941020

L13 ANSWER 12 OF 19 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 95002001 MEDLINE
DOCUMENT NUMBER: 95002001 PubMed ID: 7918499
TITLE: Coupling of calcium to the interaction of troponin I with troponin C from cardiac muscle.
AUTHOR: Liao R; Wang C K; Cheung H C
CORPORATE SOURCE: Graduate Program in Biophysical Sciences, University of Alabama at Birmingham 35294-2041.
CONTRACT NUMBER: AR25193 (NIAMS)
SOURCE: BIOCHEMISTRY, (1994 Oct 25) 33 (42) 12729-34.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199411
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941122

L13 ANSWER 13 OF 19 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 88139401 MEDLINE
DOCUMENT NUMBER: 88139401 PubMed ID: 2830278
TITLE: Troponin I enhances acidic pH-induced depression of Ca2+ binding to the regulatory sites in skeletal troponin C.
AUTHOR: el-Saleh S C; Solaro R J
CORPORATE SOURCE: Department of Physiology and Biophysics, University of Cincinnati College of Medicine, Ohio 45267.
CONTRACT NUMBER: HL 07382 (NHLBI)
HL 22231 (NHLBI)
HL 22619 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1988 Mar 5) 263 (7) 3274-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198804
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19880406

L13 ANSWER 14 OF 19 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 88050893 MEDLINE
DOCUMENT NUMBER: 88050893 PubMed ID: 3676297
TITLE: Interactions of troponin subunits: **free** energy of binary and ternary complexes.
AUTHOR: Cheung H C; Wang C K; Malik N A
CORPORATE SOURCE: Department of Biochemistry, University of Alabama at Birmingham 35924.
CONTRACT NUMBER: AM25193 (NIADDK)
SOURCE: BIOCHEMISTRY, (1987 Sep 8) 26 (18) 5904-7.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198801
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19880120

L13 ANSWER 15 OF 19 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 86085886 MEDLINE
DOCUMENT NUMBER: 86085886 PubMed ID: 3941095
TITLE: Calcium binding to the low affinity sites in troponin C induces conformational changes in the high affinity domain.
A possible route of information transfer in activation of muscle contraction.
AUTHOR: Grabarek Z; Leavis P C; Gergely J
CONTRACT NUMBER: HL-05811 (NHLBI)
HL-20464 (NHLBI)
HL-5949 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1986 Jan 15) 261 (2) 608-13.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198602
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860214

=> d l13 ibib ab kwic 11-15

L13 ANSWER 11 OF 19 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 94375482 MEDLINE
DOCUMENT NUMBER: 94375482 PubMed ID: 8089144
TITLE: NMR studies delineating spatial relationships within the cardiac troponin I-**troponin C complex**.
AUTHOR: Krudy G A; Kleerekoper Q; Guo X; Howarth J W; Solaro R J; Rosevear P R
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Texas Medical School, Houston 77225.
CONTRACT NUMBER: HL22231 (NHLBI)
HL45724 (NHLBI)
HL49934 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Sep 23) 269 (38) 23731-5.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199410
ENTRY DATE: Entered STN: 19941031
Last Updated on STN: 19941031
Entered Medline: 19941020
AB NMR spectroscopy and selective isotope labeling of both recombinant cardiac **troponin C** (cTnC3) and a truncated cardiac troponin I (**cTnI**/NH2) lacking the N-terminal 32-amino acid cardiac-specific sequence have been used to **probe** protein-protein interactions central to muscle contraction. Using [methyl-13C]Met-labeled cTnC3, all 10 cTnC Met residues of

Ca(2+)-saturated cTnC3 could be resolved in the two-dimensional heteronuclear single- and multiple-quantum coherence spectrum of the **cTnI**.cTnC **complex**. Based on the known Met assignments in cTnC3, the largest chemical shift changes were observed for Met81, Met120, Met137, and Met157. Methionines 120, 137, and 157 are all located in the C-terminal domain of cTnC. Methionine 81 is located at the N terminus of the central helix. Minimal chemical shift changes were observed for Met45, Met47, and Met103 of cTnC3 in the **cTnI**.cTnC **complex**. All 6 Met residues in [methyl13C]Met-labeled **cTnI**/NH2 could be resolved in the **cTnI**.cTnC **complex**, suggesting that both **cTnI** and cTnC form a stable homogeneous binary complex under the conditions of the NMR experiment. In the absence of added protease inhibitors in the **cTnI**.cTnC **complex**, **cTnI**/NH2 was found to undergo selective proteolysis to yield a 5.5-kDa N-terminal fragment corresponding to residues 33-80. Judging from the NMR spectra of [methyl13C]Met-labeled cTnC3, **cTnI**-(33-80) was sufficient for interaction with the C-terminal domain of cTnC in a manner identical to that observed for native **cTnI**/NH2. However, in the presence of the proteolytic fragment **cTnI**-(33-80), the chemical shift of Met81 was not perturbed from its position in **free** cTnC3. Thus, residues located C-terminal to Arg80 in **cTnI** appear to be responsible for interaction with the N-terminal half of cTnC. Taken together, these results provide strong evidence for an antiparallel arrangement for the two proteins in the troponin complex such that the N-terminal portion of **cTnI** interacts with the C-terminal domain of cTnC. This interaction likely plays a role in maintaining the stability of the **TnI**.TnC **complex**.

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AB NMR spectroscopy and selective isotope labeling of both recombinant cardiac **troponin C** (cTnC3) and a truncated cardiac troponin I (**cTnI**/NH2) lacking the N-terminal 32-amino acid cardiac-specific sequence have been used to **probe** protein-protein interactions central to muscle contraction. Using [methyl-13C]Met-labeled cTnC3, all 10 cTnC Met residues of Ca(2+)-saturated cTnC3 could be resolved in the two-dimensional heteronuclear single- and multiple-quantum coherence spectrum of the **cTnI**.cTnC **complex**. Based on the known Met assignments in cTnC3, the largest chemical shift changes were observed for Met81, Met120, Met137, and. . . terminus of the central helix. Minimal chemical shift changes were observed for Met45, Met47, and Met103 of cTnC3 in the **cTnI**.cTnC **complex**. All 6 Met residues in [methyl13C]Met-labeled **cTnI**/NH2 could be resolved in the **cTnI**.cTnC **complex**, suggesting that both **cTnI** and cTnC form a stable homogeneous binary complex under the conditions of the NMR experiment. In the absence of added protease inhibitors in the **cTnI**.cTnC **complex**, **cTnI**/NH2 was found to undergo selective proteolysis to yield a 5.5-kDa N-terminal fragment corresponding to residues 33-80. Judging from the NMR spectra of [methyl13C]Met-labeled cTnC3, **cTnI**-(33-80) was sufficient for interaction with the C-terminal domain of cTnC in a manner identical to that observed for native **cTnI**/NH2. However, in the presence of the proteolytic fragment **cTnI**-(33-80), the chemical shift of Met81 was not perturbed from its position in **free** cTnC3. Thus, residues located C-terminal to Arg80 in **cTnI** appear to be responsible for interaction with the N-terminal half of cTnC. Taken together, these results provide strong evidence for an antiparallel arrangement for the two proteins in the troponin complex such that the N-terminal portion of **cTnI** interacts with the C-terminal domain of cTnC. This interaction likely plays a role in maintaining the stability

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L13 ANSWER 12 OF 19 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 95002001 MEDLINE
DOCUMENT NUMBER: 95002001 PubMed ID: 7918499
TITLE: Coupling of calcium to the interaction of troponin I with troponin C from cardiac muscle.
AUTHOR: Liao R; Wang C K; Cheung H C
CORPORATE SOURCE: Graduate Program in Biophysical Sciences, University of Alabama at Birmingham 35294-2041.
CONTRACT NUMBER: AR25193 (NIAMS)
SOURCE: BIOCHEMISTRY, (1994 Oct 25) 33 (42) 12729-34.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199411
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941122

AB The interaction of troponin I (**CTnI**) with **troponin C** (CTnC) from bovine cardiac muscle was studied using CTnC modified at Cys35 and Cys84 with the fluorescent **probe** 2-[(4'-iodoacetamido)-anilino]naphthalene-6-sulfonic acid (CTnCIAANS). The association constant for complex formation between the two proteins was determined at 20 degrees C in 0.4 M KCl, 1 mM DTT, 1 mM EGTA, and 25 mM MOPS, pH 7.2. In the presence of EGTA, Mg2+, and Ca2+ these constants were 1.46 x 10(7), 4.1 x 10(7), and 12.7 x 10(7) M-1, respectively, with corresponding **free** energy values of -9.62, -10.23, and -10.88 kcal mol-1. The **CTnI**-CTnCIAANS **complex** was stabilized by -0.61 kcal when the two Ca/Mg sites of CTnCIAANS were saturated with Mg2+ and by -1.26 kcal when all three Ca2+ sites were occupied by Ca2+. These results suggest that calcium activation in cardiac muscle may be accompanied by a coupling **free** energy of -0.65 kcal. This value is a factor of 4 smaller than the value previously determined, using a similar method, for the (troponin I).(**troponin C**) **complex** from skeletal muscle [Wang, C.-K., & Cheung, H.C. (1985) Biophys. J.48, 727-739]. Since CTnC has only one Ca(2+)-specific site and **troponin C** from skeletal muscle has two such sites, the present result is a factor of 2 smaller than that for the skeletal complex on the basis of a single specific site. Phosphorylation of **CTnI** by 3',5'-cyclic AMP-dependent protein kinase resulted in a decrease of the association constants by a factor of 2.5-3.5.(ABSTRACT TRUNCATED AT 250 WORDS)

AB The interaction of troponin I (**CTnI**) with **troponin C** (CTnC) from bovine cardiac muscle was studied using CTnC modified at Cys35 and Cys84 with the fluorescent **probe** 2-[(4'-iodoacetamido)-anilino]naphthalene-6-sulfonic acid (CTnCIAANS). The association constant for complex formation between the two proteins was determined at 20 degrees C in. . . Mg2+, and Ca2+ these constants were 1.46 x 10(7), 4.1 x 10(7), and 12.7 x 10(7) M-1, respectively, with corresponding **free** energy values of -9.62, -10.23, and -10.88 kcal mol-1. The **CTnI**-CTnCIAANS **complex** was stabilized by -0.61 kcal when the two Ca/Mg sites of CTnCIAANS were saturated with Mg2+ and by -1.26 kcal. . . sites were occupied by Ca2+. These results suggest that calcium activation in cardiac muscle may be accompanied by a coupling **free** energy of -0.65 kcal. This value is a factor of 4 smaller than the value previously determined, using a similar method, for the (troponin I).(**troponin C**) **complex** from skeletal muscle [Wang, C.-K., & Cheung, H.C. (1985) Biophys. J.48,

727-739]. Since CTnC has only one Ca(2+)-specific site and **troponin C** from skeletal muscle has two such sites, the present result is a factor of 2 smaller than that for the skeletal complex on the basis of a single specific site. Phosphorylation of **CTnI** by 3',5'-cyclic AMP-dependent protein kinase resulted in a decrease of the association constants by a factor of 2.5-3.5.(ABSTRACT TRUNCATED AT.

L13 ANSWER 13 OF 19 MEDLINE DUPLICATE 12

ACCESSION NUMBER: 88139401 MEDLINE
DOCUMENT NUMBER: 88139401 PubMed ID: 2830278
TITLE: Troponin I enhances acidic pH-induced depression of Ca2+ binding to the regulatory sites in skeletal troponin C.
AUTHOR: el-Saleh S C; Solaro R J
CORPORATE SOURCE: Department of Physiology and Biophysics, University of Cincinnati College of Medicine, Ohio 45267.
CONTRACT NUMBER: HL 07382 (NHLBI)
HL 22231 (NHLBI)
HL 22619 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1988 Mar 5) 263 (7) 3274-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198804
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19880406

AB Inhibition of muscle force development by acidic pH is a well known phenomenon, yet the exact mechanism by which a decrease in pH inhibits the Ca2+-activated force in striated myofilaments remains poorly understood. Whether or not the deactivation by acidic pH involves direct competition between Ca2+ and protons for regulatory binding sites on fast skeletal **troponin C** (TnC) or whether other proteins in thin filament regulation are important remains unclear. We measured the effects of acidic pH on Ca2+-dependent fluorescent changes in TnC labeled with the **probe** danzylaziridine (Danz), which reports Ca2+ binding to the regulatory (Ca2+-specific) sites. Measurements were also made with TnCDanz **complexed** with the inhibitory Tn unit, **TnI**, and in the whole Tn **complex**. Our results show that a drop in pH from 7.0 to 6.5 is associated with a 1.6-fold increase in the midpoint for the relation between **free** Ca2+ and Ca2+ binding to the regulatory sites on TnCDanz. However, when TnCDanz was present in its **complex** with either **TnI** alone or with **TnI**-TnT, the increase in midpoint **free** Ca2+ was increased by 3.5-fold. We tested whether this potentiation in the effect of acidic pH on Ca2+ binding to TnC is due to a pH-induced alteration in the binding of **TnI** to TnC. A decrease in pH from 7.0 to 6.5 was associated with a halving of the affinity of **TnI** for TnC. We also **probed** the effect of acidic pH on **TnI**. This was done (i) by measuring the intrinsic fluorescence of tryptophan residues in **TnI** alone and (ii) by measuring fluorescence of **TnI** (in the Tn **complex**) labeled at Cys-133 with 5-iodoacetamidofluorescein. A drop in pH from 7.0 to 6.5 was associated with a 15% decrease in intrinsic fluorescence and with a 30% decrease in the fluorescence of the 5-iodoacetamidofluorescein **probe**. We conclude, therefore, that while protons and Ca2+ may directly affect Ca2+ binding to regulatory sites on fast skeletal TnC, the

effect of acidic pH on TnC Ca2+ binding is amplified in the **TnI**
-TnC and Tn **complexes** by a pH-related effect on the affinity of
TnI for TnC.

AB . . . not the deactivation by acidic pH involves direct competition between Ca2+ and protons for regulatory binding sites on fast skeletal **troponin C** (TnC) or whether other proteins in thin filament regulation are important remains unclear. We measured the effects of acidic pH on Ca2+-dependent fluorescent changes in TnC labeled with the **probe** danzylaziridine (Danz), which reports Ca2+ binding to the regulatory (Ca2+-specific) sites. Measurements were also made with TnCDanz **complexed** with the inhibitory Tn unit, **TnI**, and in the whole Tn **complex**. Our results show that a drop in pH from 7.0 to 6.5 is associated with a 1.6-fold increase in the midpoint for the relation between **free** Ca2+ and Ca2+ binding to the regulatory sites on TnCDanz. However, when TnCDanz was present in its **complex** with either **TnI** alone or with **TnI**-TnT, the increase in midpoint **free** Ca2+ was increased by 3.5-fold. We tested whether this potentiation in the effect of acidic pH on Ca2+ binding to TnC is due to a pH-induced alteration in the binding of **TnI** to TnC. A decrease in pH from 7.0 to 6.5 was associated with a halving of the affinity of **TnI** for TnC. We also **probed** the effect of acidic pH on **TnI**. This was done (i) by measuring the intrinsic fluorescence of tryptophan residues in **TnI** alone and (ii) by measuring fluorescence of **TnI** (in the Tn **complex**) labeled at Cys-133 with 5-iodoacetamidofluorescein. A drop in pH from 7.0 to 6.5 was associated with a 15% decrease in intrinsic fluorescence and with a 30% decrease in the fluorescence of the 5-iodoacetamidofluorescein **probe**. We conclude, therefore, that while protons and Ca2+ may directly affect Ca2+ binding to regulatory sites on fast skeletal TnC, the effect of acidic pH on TnC Ca2+ binding is amplified in the **TnI** -TnC and Tn **complexes** by a pH-related effect on the affinity of **TnI** for TnC.

L13 ANSWER 14 OF 19 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 88050893 MEDLINE
DOCUMENT NUMBER: 88050893 PubMed ID: 3676297
TITLE: Interactions of troponin subunits: **free** energy of binary and ternary complexes.
AUTHOR: Cheung H C; Wang C K; Malik N A
CORPORATE SOURCE: Department of Biochemistry, University of Alabama at Birmingham 35924.
CONTRACT NUMBER: AM25193 (NIADDK)
SOURCE: BIOCHEMISTRY, (1987 Sep 8) 26 (18) 5904-7.
Journal code: A0G; 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198801
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19880120

AB We have determined the **free** energy of formation of the binary **complexes** formed between skeletal **troponin C** and troponin T (TnC.TnT) and between troponin T and troponin I (TnT. **TnI**). This was accomplished by using TnC fluorescently modified at Cys-98 with N-(iodoacetyl)-N'-(5-sulfo-1-naphthyl)ethylenediamine for the first **complex** and **TnI** labeled at Cys-133 with the same **probe** for the other complex. The **free** energy of the ternary **complex** formed between **troponin C** and the binary **complex** TnT.**TnI** [TnC.(TnT.**TnI**

)] was also measured by monitoring the emission of 5-(iodoacetamido)eosin attached to Cys-133 of the troponin I in TnT.**TnI**. The **free** energies were -9.0 kcal.mol-1 for TnC.TnT, -9.2 kcal.mol-1 for TnT.**TnI**, and -8.7 kcal.mol-1 for TnC.(TnT.**TnI**). In the presence of Mg2+ the **free** energies of TnC.TnT and TnC.(TnT.**TnI**) were -10.3 and -10.9 kcal.mol-1, respectively; in the presence of Ca2+ the corresponding **free** energies were -10.6 and -13.5 kcal.mol-1. Mg2+ and Ca2+ had negligible effect on the **free** energy of TnT.**TnI**. From these results the **free** energies of the formation of troponin from the three subunits were found to be -16.8 kcal.mol-1, -18.9 kcal.mol-1, and -21.6 kcal.mol-1 in the presence of EGTA, Mg2+, and Ca2+, respectively. Most of the **free** energy decrease caused by Ca2+ binding to the Ca2+-specific sites is derived from stabilization of the **TnI**-TnC linkage.(ABSTRACT TRUNCATED AT 250 WORDS)

TI Interactions of troponin subunits: **free** energy of binary and ternary complexes.

AB We have determined the **free** energy of formation of the binary **complexes** formed between skeletal **troponin C** and troponin T (TnC.TnT) and between troponin T and troponin I (TnT.**TnI**). This was accomplished by using TnC fluorescently modified at Cys-98 with N-(iodoacetyl)-N'-(5-sulfo-1-naphthyl)ethylenediamine for the first **complex** and **TnI** labeled at Cys-133 with the same **probe** for the other complex. The **free** energy of the ternary **complex** formed between **troponin C** and the binary **complex** TnT.**TnI** [TnC.(TnT.**TnI**)] was also measured by monitoring the emission of 5-(iodoacetamido)eosin attached to Cys-133 of the troponin I in TnT.**TnI**. The **free** energies were -9.0 kcal.mol-1 for TnC.TnT, -9.2 kcal.mol-1 for TnT.**TnI**, and -8.7 kcal.mol-1 for TnC.(TnT.**TnI**). In the presence of Mg2+ the **free** energies of TnC.TnT and TnC.(TnT.**TnI**) were -10.3 and -10.9 kcal.mol-1, respectively; in the presence of Ca2+ the corresponding **free** energies were -10.6 and -13.5 kcal.mol-1. Mg2+ and Ca2+ had negligible effect on the **free** energy of TnT.**TnI**. From these results the **free** energies of the formation of troponin from the three subunits were found to be -16.8 kcal.mol-1, -18.9 kcal.mol-1, and -21.6 kcal.mol-1 in the presence of EGTA, Mg2+, and Ca2+, respectively. Most of the **free** energy decrease caused by Ca2+ binding to the Ca2+-specific sites is derived from stabilization of the **TnI**-TnC linkage.(ABSTRACT TRUNCATED AT 250 WORDS)

L13 ANSWER 15 OF 19 MEDLINE DUPLICATE 14

ACCESSION NUMBER: 86085886 MEDLINE
DOCUMENT NUMBER: 86085886 PubMed ID: 3941095
TITLE: Calcium binding to the low affinity sites in troponin C induces conformational changes in the high affinity domain.
A possible route of information transfer in activation of muscle contraction.
AUTHOR: Grabarek Z; Leavis P C; Gergely J
CONTRACT NUMBER: HL-05811 (NHLBI)
HL-20464 (NHLBI)
HL-5949 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1986 Jan 15) 261 (2) 608-13.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198602
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860214

AB Residues 89-100 of **troponin C** (C89-100) and 96-116 of

troponin I (I96-116) interact with each other in the troponin complex (Dalgarno, D.C., Grand, R.J.A., Levine, B.A. Moir, A., J.G., Scott, G.M.M., and Perry, S.V. (1982) FEBS Lett. 150, 54-58) and are necessary for the Ca2+ sensitivity of actomyosin ATPase (Syska, H., Wilkinson, J.M., Grand, R.J.A., and Perry, S.V. (1976) Biochem. J. 153, 375-387 and Grabarek, Z., Drabikowski, W., Leavis, P.C., Rosenfeld, S.S., and Gergely, J. (1981) J. Biol. Chem. 256, 13121-13127). We have studied Ca2+-induced changes in the region C89-100 by monitoring the fluorescence of **troponin C** (TnC) labeled at Cys-98 with 5-(iodoacetamidoethyl)aminonaphthalene-1-sulfonic acid. Equilibrium titration of the labeled TnC with Ca2+ indicates that the **probe** is sensitive to binding to both classes of sites in **free** TnC as well as in its **complex** with **TnI**. When Mg2 X TnC is mixed with Ca2+ in a stopped flow apparatus, there is a rapid fluorescence increase related to Ca2+ binding to the unoccupied sites I and II followed by a slower increase (k = 9.9 s-1) that represents Mg2+-Ca2+ exchange at sites III and IV. In the TnC X **TnI complex**, the fast phase is much larger and the Mg2+-Ca2+ exchange at sites III and IV results in a small decrease rather than an increase in the fluorescence of the **probe**. The possibility is discussed that the fast change in the environment of Cys-98 upon Ca2+ binding to sites I and II may be instrumental in triggering activation of the thin filament by facilitating a contact between C89-100 and I96-116.

AB Residues 89-100 of **troponin C** (C89-100) and 96-116 of troponin I (I96-116) interact with each other in the troponin complex (Dalgarno, D.C., Grand, R.J.A., Levine,. . . (1981) J. Biol. Chem. 256, 13121-13127). We have studied Ca2+-induced changes in the region C89-100 by monitoring the fluorescence of **troponin C** (TnC) labeled at Cys-98 with 5-(iodoacetamidoethyl)aminonaphthalene-1-sulfonic acid. Equilibrium titration of the labeled TnC with Ca2+ indicates that the **probe** is sensitive to binding to both classes of sites in **free** TnC as well as in its **complex** with **TnI**. When Mg2 X TnC is mixed with Ca2+ in a stopped flow apparatus, there is a rapid fluorescence increase related. . . a slower increase (k = 9.9 s-1) that represents Mg2+-Ca2+ exchange at sites III and IV. In the TnC X **TnI complex**, the fast phase is much larger and the Mg2+-Ca2+ exchange at sites III and IV results in a small decrease rather than an increase in the fluorescence of the **probe**. The possibility is discussed that the fast change in the environment of Cys-98 upon Ca2+ binding to sites I and. . .

=> d l13 ibib 16-19

L13 ANSWER 16 OF 19 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 86281164 MEDLINE
DOCUMENT NUMBER: 86281164 PubMed ID: 2942677
TITLE: Stimulation of cardiac myofilament force, ATPase activity and troponin C Ca++ binding by bepridil.
AUTHOR: Solaro R J; Bousquet P; Johnson J D
CONTRACT NUMBER: AM 33727 (NIADDK)
HL-22231 (NHLBI)
SOURCE: JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, (1986 Aug) 238 (2) 502-7.
Journal code: JP3; 0376362. ISSN: 0022-3565.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198609
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860917

L13 ANSWER 17 OF 19 MEDLINE DUPLICATE 16
ACCESSION NUMBER: 86077956 MEDLINE
DOCUMENT NUMBER: 86077956 PubMed ID: 4074834
TITLE: Energetics of the binding of calcium and troponin I to troponin C from rabbit skeletal muscle.
AUTHOR: Wang C K; Cheung H C
CONTRACT NUMBER: AM25193 (NIADDK)
SOURCE: BIOPHYSICAL JOURNAL, (1985 Nov) 48 (5) 727-39.
Journal code: A5S; 0370626. ISSN: 0006-3495.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198602
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860218

L13 ANSWER 18 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1983:239114 BIOSIS
DOCUMENT NUMBER: BA75:89114
TITLE: FLUORESCENCE ENERGY TRANSFER STUDIES OF SKELETAL TROPONIN C

PROXIMITY BETWEEN METHIONINE 25 AND CYSTEINE 98.
AUTHOR(S): CHEUNG H C; WANG C-K; GARLAND F
CORPORATE SOURCE: BIOPHYSICS SECTION, DEP. BIOMATH., UNIV. ALABAMA BIRMINGHAM, BIRMINGHAM, ALA. 35294.
SOURCE: BIOCHEMISTRY, (1982) 21 (21), 5135-5142.
CODEN: BICHAW. ISSN: 0006-2960.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L13 ANSWER 19 OF 19 MEDLINE DUPLICATE 17
ACCESSION NUMBER: 81191909 MEDLINE
DOCUMENT NUMBER: 81191909 PubMed ID: 7228870
TITLE: A new heterobifunctional cross-linking reagent for the study of biological interactions between proteins. II. Application to the troponin C-troponin I interaction.
AUTHOR: Chong P C; Hodges R S
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1981 May 25) 256 (10) 5071-6.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198107
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19810720

=> d l13 ibib ab kwic 16,17,19

L13 ANSWER 16 OF 19 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 86281164 MEDLINE
DOCUMENT NUMBER: 86281164 PubMed ID: 2942677
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AUTHOR: Solaro R J; Bousquet P; Johnson J D
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Journal code: JP3; 0376362. ISSN: 0022-3565.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198609
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860917

AB We report that bepridil, a Ca++ channel blocker and calmodulin antagonist, which has been shown to enter myocytes, stimulates the mechanical and biochemical activity of cardiac myofilaments. Bepridil increased significantly the level of Ca++-dependent actomyosin Mg++-ATPase activity of myofibrils and the submaximal force developed by chemically skinned trabeculae of pig heart. In the range of concentrations (10-100 microM) in which bepridil showed this stimulatory activity, diltiazem and verapamil were without effect. The effect of bepridil on myofilament force and ATPase activity was higher at relatively low **free** Ca++ concentrations, and myofibrils lacking troponin-tropomyosin were not affected by bepridil. Associated with the stimulation of force and ATPase activity by bepridil was an increase in the amounts of Ca++ **bound** to **troponin C** (TnC). That bepridil stimulates TnC Ca++ binding was also shown in experiments using pure TnC labeled with 2-(4'-iodoacetamidoanilo)naphthalene-6-sulfonic acid, a fluorescent **probe** that reports Ca++ bound to the single "regulatory" site. Effects of bepridil on the fluorescence of a felodipine-cardiac TnC complex indicate that bepridil binds to TnC over the same range of doses where it affects myofilament activity. Our results indicate that the inotropic action of bepridil may result from a net response of heart cells to influences on the delivery of Ca++ to the myofilaments and their response to Ca++.

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L13 ANSWER 17 OF 19 MEDLINE DUPLICATE 16
ACCESSION NUMBER: 86077956 MEDLINE
DOCUMENT NUMBER: 86077956 PubMed ID: 4074834
TITLE: Energetics of the binding of calcium and troponin I to troponin C from rabbit skeletal muscle.
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SOURCE: BIOPHYSICAL JOURNAL, (1985 Nov) 48 (5) 727-39.
Journal code: A5S; 0370626. ISSN: 0006-3495.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 198602
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860218

AB We determined the **free** energy of interaction between rabbit skeletal troponin I (**TNI**) and **troponin C** (TNC) at 10 degrees and 20 degrees C with fluorescently labeled proteins. The sulfhydryl **probe** 5-iodoacetamidoeosin (IAE) was attached to cysteine (Cys)-98 of TNC and to Cys-133 of **TNI**, and each of the labeled proteins was titrated with the other unlabeled protein. The association constant for formation of the **complex** between labeled TNC (TNC*) and **TNI** was 6.67 X 10(5) M-1 in 0.3 M KCl, and pH 7.5 at 20 degrees C. In the presence of bound Mg2+, the binding constant increased to 4.58 X 10(7) M-1 and in the presence of excess of Ca2+, the association constant was 5.58 X 10(9) M-1. Very similar association constants were obtained when labeled **TNI** was titrated with unlabeled TNC. The energetics of Ca2+ binding to TNC* and the **complex TNI** X TNC* were also determined at 20 degrees C. The two sets of results were used to separately determine the coupling **free** energy for binding **TNI** and Mg2+, or Ca2+ to TNC. The results yielded a total coupling **free** energy of -5.4 kcal. This **free** energy appeared evenly partitioned into the two species: **TNI** X TNC(Mg)2 or **TNI** X TNC(Ca)2, and **TNI** X TNC(Ca)4. The first two species were each stabilized by -2.6 kcal, with respect to the Ca2+ **free TNI** X TNC **complex**, and **TNI** X TNC(Ca)4 was stabilized by -2.8 kcal, respect to **TNI** X TNC(Ca)2 or **TNI** X TNC(Mg)2. The coupling **free** energy was shown to produce cooperatively **complexes** formed between **TNI** and TNC in which the high affinity sites were initially saturated as a function of **free** Ca2+ to yield **TNI** X TNC(Ca)4. This saturation occurred in the **free** Ca2+ concentration range 10(-7) to 10(-5) M. The cooperative strengthening of the linkage between **TNI** and TNC induced by Ca2+ binding to the Ca2+-specific sites of TNC may have a direct relationship to activation of actomyosin ATPase. The nature of the forces involved in the Ca2+-induced strengthening of the complex is discussed.

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L13 ANSWER 19 OF 19 MEDLINE DUPLICATE 17
ACCESSION NUMBER: 81191909 MEDLINE
DOCUMENT NUMBER: 81191909 PubMed ID: 7228870
TITLE: A new heterobifunctional cross-linking reagent for the study of biological interactions between proteins. II. Application to the troponin C-troponin I interaction.
AUTHOR: Chong P C; Hodges R S
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1981 May 25) 256 (10) 5071-6.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198107
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19810720

AB A simple chromatographic procedure using DEAE-Sephadex has been established to isolate the troponin I-**troponin C** **complex** from **unbound** troponin I (**TnI**) and **troponin C** (TnC). A 1:1 **complex** can be formed between bovine cardiac carboxamidomethylated troponin I and rabbit skeletal **troponin C**. The formation of the **complex** is calcium dependent. It is stable to DEAE-chromatography in 6 M urea, 3 mM Ca2+ and can be dissociated on DEAE-chromatography in the presence of 6 M urea, 1 mM ethylene glycol bis(beta-aminoethyl ether)-N,N,N',N'-tetraacetic acid. TnC was modified with the photoaffinity **probe** AGTC (N-(4-azidobenzoyl-[2-3H]glycyl)-S-(2-thiopyridyl)-cysteine) at its single cysteine residue (position 98). Photolysis of the CM (carboxamidomethylated)-**TnI**-AGC-TnC **complex** resulted in the formation of a covalently linked 1:1 complex. The isolated covalently linked complex could be treated with dithiothreitol to reduce the disulfide bond between N-(4-azidobenzoyl-[2-3H]glycyl)-cysteine (AGC) and TnC to complete the transfer of the radiolabeled AGC from cysteine 98 on TnC to CM-**TnI**. The CM-**TnI**-AGC was isolated from TnC on DEAE-chromatography in 6 M urea, 1 mM EGTA, 1 mM dithiothreitol buffer.
The formation of the covalent bond between the photoaffinity **probe** and **TnI** indicates the close proximity of **TnI** to cysteine 98 on the TnC. These results demonstrate the general utility of the new heterobifunctional cross-linking reagent to study protein interactions.
AB A simple chromatographic procedure using DEAE-Sephadex has been established to isolate the troponin I-**troponin C** **complex** from **unbound** troponin I (**TnI**) and **troponin C** (TnC). A 1:1 **complex** can be formed between bovine cardiac carboxamidomethylated troponin I and rabbit skeletal **troponin C**. The formation of the **complex** is calcium dependent. It is stable to DEAE-chromatography in 6 M urea, 3 mM Ca2+ and can be dissociated on. . . in the presence of 6 M urea, 1 mM ethylene glycol bis(beta-aminoethyl ether)-N,N,N',N'-tetraacetic acid. TnC was modified with the photoaffinity **probe** AGTC (N-(4-azidobenzoyl-[2-3H]glycyl)-S-(2-thiopyridyl)-cysteine) at its single cysteine residue (position 98). Photolysis of the CM (carboxamidomethylated)-**TnI**-AGC-TnC **complex** resulted in the formation of a covalently linked 1:1 complex. The isolated covalently linked complex could be treated with dithiothreitol. . . bond between N-(4-azidobenzoyl-[2-3H]glycyl)-cysteine (AGC) and TnC to complete the transfer of the radiolabeled AGC from cysteine 98 on TnC to CM-**TnI**. The CM-**TnI**-AGC was isolated from TnC on DEAE-chromatography in 6 M urea, 1 mM EGTA, 1 mM dithiothreitol buffer. The formation of the covalent bond between the photoaffinity **probe**

and **TnI** indicates the close proximity of **TnI** to cysteine 98 on the TnC. These results demonstrate the general utility of the new heterobifunctional cross-linking reagent to study. . .

=> d his

(FILE 'HOME' ENTERED AT 13:32:07 ON 18 SEP 2001)

FILE 'MEDLINE, EMBASE, SCISEARCH, BIOSIS' ENTERED AT 13:32:26 ON 18 SEP 2001

L1 8664 S (TROPONIN C) OR CTNI OR TNI
L2 105 S BINARY (5A) ((TROPONIN C) OR CTNC OR TNC)
L3 63 S TERNARY (10A) ((TROPONIN C) OR CTNC OR TNC) (10A) ((TROPONIN
L4 105 S L1 (6P) L2
L5 63 S L1 (6P) L3
L6 964 S L1 (6P) (ANTIBOD? OR RECEPTOR? OR PROBE? OR ANTITROPONIN OR A
L7 45 S L6 AND L4
L8 20 S L6 AND L5
L9 15 DUP REM L7 (30 DUPLICATES REMOVED)
L10 6 DUP REM L8 (14 DUPLICATES REMOVED)
L11 189 S L1 (6A) (BOUND OR COMPLEX?) AND (UNBOUND OR FREE OR UNCOMPLEX
L12 61 S L11 AND L6
L13 19 DUP REM L12 (42 DUPLICATES REMOVED)

=> s l1 and (sensitiv? (3a) antibod?)

L14 1 L1 AND (SENSITIV? (3A) ANTIBOD?)

=> d l14 kwic

L14 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2001 BIOSIS
AB. . . with other RLCs, VRLC and MRLC may be phosphorylated at Ser-11 by an endogenous kinase. All myosin light chains, like **troponin C** and calmodulin, contain four homologous regions, I to IV, each of which contains a twelve-residue potential Ca2+-binding loop flanked on.
. .
IT Miscellaneous Descriptors
MERCENARIA-MERCENARIA MACROCALLISTA-NIMBOSA MUSCLE CONTRACTION REGULATION CALCIUM **SENSITIVE** BINDING SITES **ANTIBODIES** PROTEIN CHEMISTRY METHODS

=> d l14 ibib ab kwic 1

L14 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1991:455852 BIOSIS
DOCUMENT NUMBER: BA92:100632
TITLE: AMINO ACID SEQUENCES OF MYOSIN ESSENTIAL AND REGULATORY LIGHT CHAINS FROM TWO CLAM SPECIES COMPARISON WITH OTHER MOLLUSCAN MYOSIN LIGHT CHAINS.
AUTHOR(S): BAROUCH W W; BREESE K E; DAVIDOFF S-A; LESZYK J; SZENT-GYORGYI A G; THEIBERT J L; COLLINS J H
CORPORATE SOURCE: MEDICAL BIOTECHNOL. CENTER, MARYLAND BIOTECHNOL. INST., UNIV. MARYLAND, BALTIMORE, MD. 21201.
SOURCE: J MUSCLE RES CELL MOTIL, (1991) 12 (4), 321-332. CODEN: JMRMD3. ISSN: 0142-4319.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB We have determined the amino acid sequences of the essential light chains (ELC) and regulatory light chains (RLC) of myosin from two species of clam, Mercenaria mercenaria and Macrocallista nimbosa, using protein chemistry methods. The N-termini of all four proteins were blocked, and sequencing was carried out on various chemically and enzymatically produced peptide fragments. Cleavage of either Mercenaria RLC (MRLC) or Macrocallista RLC (VLC) at its 3 Arg yielded four peptides, three of which could not be sequenced directly, due to an N-terminal blocking group and 2 Arg-Gln bonds in these proteins. The fourth peptide was partially and specifically cleaved at an unusually reactive residue, Met-64, which is invariant in all known RLC sequences. A comparison of all available molluscan ELC and RLC sequences was carried out in search of clues to functionally important features of these proteins in muscles which are regulated by a Ca2+-sensitive myosin. By analogy with other RLCs, VRLC and MRLC may be phosphorylated at Ser-11 by an endogenous kinase. All myosin light chains, like **troponin C** and calmodulin, contain four homologous regions, I to IV, each of which contains a twelve-residue potential Ca2+-binding loop flanked on either side by a pair of helices. All RLCs, including those from Ca2+-insensitive myosins, contain a divalent cation-binding site in region I. Clam and other molluscan ELCs contain a single Ca2+-binding site in region III. This site is present only in the ELCs of myosins that are regulated by direct binding of Ca2+. The ELC site III is likely to play a key role in the regulation of molluscan muscle contraction.

AB. . . with other RLCs, VRLC and MRLC may be phosphorylated at Ser-11 by an endogenous kinase. All myosin light chains, like **troponin C** and calmodulin, contain four homologous regions, I to IV, each of which contains a twelve-residue potential Ca2+-binding loop flanked on.

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MERCENARIA-MERCENARIA MACROCALLISTA-NIMBOSA MUSCLE CONTRACTION REGULATION CALCIUM **SENSITIVE** BINDING SITES **ANTIBODIES** PROTEIN CHEMISTRY METHODS